The Southern Surgeon

Subscription in the United States, $5.00

Vol. XI, No. 11

Copyright 1942 by
The Southern Surgeon Publishing Co.
November, 1942

INJURIES OF THE SPINAL CORD AND CAUDA EQUINA

COBB PILCHER, M. D.
Nashville

LIKE Humpty-Dumpty, the spinal cord can never be put together again! At first glance this gives the subject a hopeless cast and suggests that this paper might better be left unread. However, such a defeatist attitude would neglect the possibilities of prevention of further injury of associated bony injuries, of palliative therapeutic methods, of avoidance of complications and of adequate diagnosis, with its resultant correct choice of therapy and accuracy of prognosis.

In no field of surgery are the results perfect. Only by continuing study of our unsolved and seemingly impossible problems may further progress be made. To the neurosurgeon, this is not a strange difficulty, for he has seen his relatively young specialty grow and improve by leaps and bounds, even in very recent years. Only fifty odd years ago, Horsley was first removing a spinal cord tumor, McEwen was treating brain abscesses successfully for the first time and Keen was removing a huge brain tumor from a patient whom he was able to report as well thirty years later.

Thus, in this consideration of spinal cord injuries, we must not only face the hopelessness of many cases, but we must also discuss the mechanisms of such injuries, associated damage that may occur, sequellae which may follow and complications which may be preventable or amenable to treatment. We shall be concerned with acute injuries and time will not permit discussion of the more chronic and recurrent disorders such as herniation of the nucleus pulposus following rupture of an intervertebral disc. Further, any such discussion would be repetitious, since I spoke on that subject in Kansas City only a few months ago.

From the Department of Surgery, Vanderbilt University School of Medicine.

Read before the Kansas City Southeast Clinical Society, Kansas City, Mo., Oct. 7, 1942.

Injuries of the spinal cord are almost indissolubly linked with injuries of the vertebral column and in any consideration of the former we must also take cognizance of the latter. The vertebral column has two essential functions: to bear weight and to enclose and protect the spinal cord, the cauda equina and the spinal nerves. Trauma may result in its failure in one or both of these functions.

The spine, of which we are accustomed to think as a series of bones set one upon the other, might also be considered as a series of joints separated by the vertebral bones. For the mobility of these articulations influences in no small way the resultant effects of trauma. Thus fractures are much more common in the highly movable cervical and lumbar regions than in the dorsal spine whose mobility is greatly limited by the thoracic cage. (From this point of view, the lowest two dorsal vertebrae should be considered along with the lumbar spine.) And fractures of the sacrum are relatively rare.

Similarly, the normal curvatures of the spine, anterior in the cervical and lumbar regions, posterior in the dorsal, play an important role, as will be seen.

The spinal cord is suspended in the cerebrospinal fluid of the relatively spacious dural canal by a series of dentate ligaments, delicate filaments of arachnoidal membrane. Anterior and posterior nerve roots arise above their foramina of emergence and course downward for a variable distance before passing through the dura and, subsequently, through the intervertebral foramina. The cord terminates at the level of the upper border of the second lumbar vertebra in the cauda equina, whose nerves float free in the cerebrospinal fluid.

It is a remarkable fact that, although the cord will tolerate gradual compression of high degree (as by tumor) for years without permanent loss of function, recovery from sudden contusion or compression rarely, if ever, occurs. From preliminary results of animal experiments by Dr. George Duncan and myself, it may be stated that the spinal cord probably has a clean-cut threshold of trauma which can be tolerated without permanent loss of function and that this threshold represents a very mild degree of trauma. In other words, the cord is exceedingly susceptible to injury and, when injury results in actual destruction of cells or fibers, regeneration of these structures never occurs. It therefore follows that treatment must be directed toward (1) prevention of further injury, (2) treatment of concomitant injuries to bones and joints, (3) prevention of complications and (4) relief of suffering. In order intelligently to carry out such therapy, the surgeon must

analyze each case in terms of the mechanism and nature of the damage which the nervous elements may have sustained.

Mechanisms

Trauma may injure the spinal cord and its subsidiary nerves in a number of different ways.

1. *Contusion and laceration.* Injuries of this type result in most cases from fractures or dislocations of the spine or both. Accompanying a high percentage of fractures are displacements of the vertebral bones resulting from tearing of ligaments and rupture of joints. Such injuries are usually produced by indirect force, such as violent flexion, diving accidents or crushing blows from above. Bodies of the vertebrae are much more often fractured than other parts and the "triangulation" fracture, with narrowing of the anterior border of the vertebral body and resulting localized kyphotic deformity is the most common type. The most frequent dislocation is displacement of an upper vertebra anteriorly on the one beneath it. Such injuries are most common in the lower cervical and the dorso-lumbar regions. Fractures of the posterior arches and laminae are much less common, but nearly always result in injury of the cord.

Fracture-dislocations of the type described may occur without evidence of accompanying cord injury, but in the majority of cases, the cord is contused or stretched or lacerated by displaced fragments of bone. Even if no apparent injury of the cord has occurred, marked impingement upon the spinal canal may be demonstrable in roentgenograms and delayed neurologic symptoms may arise.

2. *Hematoma.* Intraspinal vessels may be lacerated and extensive bleeding may occur, with resulting clot-formation. This bleeding may be extradural, subarachnoid, or intramedullary. Extradural and subarachnoid bleeding seldom, if ever, cause serious compression of the cord, for there is adequate space, both outside and inside the dura, for extravasated blood to infiltrate in a longitudinal direction. Further, the low venous pressure in thin, collapsible intraspinal veins results in cessation of bleeding long before dangerous compression occurs.

On the other hand intramedullary hemorrhage, or hematomyelia, may cause serious cord destruction by clot-formation within the spinal cord itself. Such bleeding may result from direct contusion or from vascular changes such as sudden violent strains or changes in atmospheric pressure (as in caisson disease).

3. *Concussion of the cord.* In some cases, spinal cord lesions occur following injury when there are no demonstrable fractures of dislocations. In most such instances, it is probable that there has been

a "whip-snapping" dislocation, which has reduced itself "on the rebound," so to speak. In that event, the lesion is in reality a contusion.

However, in some cases, it has been shown that no abnormality of bones or joints had existed, but a complete transverse loss of cord function was present. Petecchial hemorrhages and swelling of the cord may be present, but in other cases, the cord may be entirely normal to microscopic examination. The mechanism of the functional loss in these cases is unknown. It may be only temporary, but more often is permanent.

4. *Penetrating wounds.* Laceration of the cord by bullets, shell-fragments or knife blades is uncommon in civilian life, but is frequently encountered in warfare. Lacerations by bullets or knives are usually clean-cut and may spare portions of the cord. Missiles may be lodged in cord, canal or bone. The factor of potential infection is superimposed upon other problems and may alter therapeutic decisions.

5. *Late post-traumatic disorders.* Even in cases having no immediate neurologic disturbances, late changes may occur. Kyphoses resulting from fractures may exhibit gradually increasing deformity with slow cord compression. Injured leptomeningeal membranes may slowly heal with the formation of contracting arachnoidal adhesions or cysts. Late development of severe nerve root pain is not uncommon in such cases.

Symptomatology

In the presence of all spinal injuries, not only the presence, but the time of development of cord symptoms is of great importance. Instantaneous paralysis is likely to be permanent, whereas slowly developing symptoms may be temporary or remediable. The nature and extent of the neurologic loss will depend, of course, on the completeness of the cord injury and upon its level.

Complete transverse lesions. Acute transverse loss of cord function is manifested by complete flaccid paralysis and loss of sensation below the level of the injury. All reflexes are lost. Bladder and rectal musculature and their respective sphincters are paralyzed.

After weeks or months, spasticity with overactive reflexes may gradually develop. Mass reflexes (slow or violent muscular contractions following skin irritation) appear. The sphincters become spastic, but relax under pressure, so that the bladder may empty itself spontaneously when a certain degree of distension is reached. These changes result from functioning of reflex arcs below the level of injury and do not themselves indicate any degree of recovery of the damaged area of the cord.

It is impossible here to review the neurologic findings in injuries at all levels of the spine. A few important points may be mentioned, however.

Complete transverse lesions above the fourth cervical segment are almost uniformly immediately fatal, due to paralysis of the muscles of respiration. Below this level, the diaphragm continues to function.

With high cervical lesions, a frequent symptom with grave prognostic significance is irregularly high fever.

Lesions at the fifth and sixth cervical levels result in paralysis of the triceps muscles, but retention of power in the biceps. Such patients characteristically lie with their elbows flexed and cannot extend them.

At the eighth cervical or first thoracic level, there is a Horner's syndrome; contraction of the pupils, enophthalmos and loss of sweating on the face.

At the tenth dorsal segment, Beevor's sign will be elicited by having the patient raise his head from the pillow. The umbilicus will move upward, due to paralysis of only the lower half of the rectus abdominalis muscles.

Lesions of the terminal portion of the cord nearly always involve lower lumbar and all sacral segments. Sacral without lumbar involvement suggests that the lesion is in the lower cauda equina.

Incomplete cord injuries. Partial functional loss in the cord may be very slight or very extensive. Sometimes careful examination of a supposedly complete transverse lesion will reveal a little remaining sensation in the lower sacral distribution.

Clean-cut hemisection of the cord, with true Brown-Sequard syndrome, is very rare, but bullets and knives may produce essentially unilateral damage. In such instances, there is nearly always some dissociation of function, with motor paralysis on the side of the injury and loss of pain and temperature sensibility on the contralateral side.

Additional examinations. Local pain in the region of a spinal fracture is often present, but, in many cases, no pain is felt. Bony deformities, especially kyphoses may be visible or palpable. Roentgenograms should be made in all cases (see under "Treatment" below), but the cord may be severely damaged in the absence of demonstrable bony abnormality.

Spinal puncture is of great importance, because of two examinations: first, for the presence and amount of blood in the spinal fluid and second for evidence of subarachnoid block. Some gross or

microscopic blood is present in most cases and its amount is roughly proportional to the extent of gross contusion or laceration of the cord, although not necessarily to the degree of loss of neurologic function.

The Queckenstedt test for cerebrospinal fluid block is carried out by measuring the pressure of the fluid through a spinal puncture needle inserted below the injury. When the jugular veins are compressed, there will be a prompt rise in spinal fluid pressure, providing the spinal canal is unobstructed. In the presence of a block no rise in pressure occurs, or, if the block is partial, the pressure may increase very slowly, and fall only gradually after release of the jugular compression.

Pitfalls. Two sources of error must be avoided. First, an unconscious patient who has received a head injury may also have an easily overlooked spinal injury. If any doubt exists, if movements of extremities are not free and equal, if reflexes are abolished, the patient should be treated as a spinal injury until all doubt is dispelled.

Second, I have seen two cases of hysterical paraplegia following apparent spinal injuries. In both, there was apparently complete loss of motor power and sensation below a given level, yet the reflexes were all normal and there was no disturbance of bladder function. Such a picture is not compatible with an organic lesion.

Treatment of Closed Injuries

Handling and transportation. All neck and back injuries, whether or not obvious evidence of cord damage is present, should be handled with greatest possible care. Movement should be minimal and should be carried out by *rolling* the patient, without either twisting or bending the spine. In the absence of a stretcher, the patient may be bound to a broad board.

Some writers insist that these cases should be transported in a face-down position, but I do not believe this is essential in any case. In the presence of cervical injury, it would involve twisting the injured spine and should never be employed.

Roentgenograms should be made while the patient is still on the stretcher and should involve a minimum of movement.

Operative treatment. The question of the wisdom of operation in cases of cord injury has long been a controversal one. Some surgeons feel that nearly all cases should be given whatever hope laminectomy may offer—a sort of "nothing-to-lose" attitude. Others believe equally strongly that operation is never of benefit. However, if we analyze the question in the light of existing knowledge and

experience, it is apparent that, like all generalizations, neither of these viewpoints is tenable.

In so far as spinal cord function is concerned, the question of operation should be decided in each case in the light of two established facts.

First, recovery from a complete transverse cord injury which takes place instantaneously never occurs.*

Second, the only thing which operation can accomplish is decompression.

It is therefore apparent that operative therapy should be employed *only* in cases in which the cord injury is incomplete or delayed and in which compression still exists. Persistence of compression may be established by visualization of displaced bone impinging upon the canal in the roentgenograms or by demonstration of a spinal fluid block by the Queckenstedt test, or by both.

Even when these criteria are satisfied, however, operation is not always indicated. For bony protrusion into the canal may be safely reducible in some cases by manipulative methods.

With few exceptions, the cases which offer the greatest hope as a result of operation are those in which there is a progression of symptoms.

In this connection, there arises the question of optimum time for operation. In my opinion, it should be undertaken, if at all, at the earliest feasible moment,** for its sole purpose is to relieve compression and thereby prevent further damage to the cord.

In addition to the indications for operative interference just mentioned, which are designed to relieve already existing compression, operation is also clearly indicated in other cases in order to make manipulative reduction of spinal dislocations safe. Such cases are those in which there are comminuted fractures of the posterior arches and laminae and those in which the articular processes are "locked," rendering primary reduction impossible.

In all cases, the operation should consist of simple, wide laminectomy. Great care must be taken not to drive bone fragments into the canal. Separation of bone from muscle must often be done by sharp dissection instead of by forcible use of the periosteal elevator. The dura should be opened only if the presence of hematomyelia is strongly suspected.

*Elsberg states that some cases of "concussion" of the cord may show recovery in a few days or weeks, but I have never seen this occur when the lesion was both complete and instantaneous.

**Naturally, shock must be combatted by immediate transfusion and other injuries must receive consideration.

Reduction of fracture-dislocations. The reduction of bony deformities must be undertaken not only to restore the continuity of the spinal canal, as mentioned above, but also to restore the alignment of the vertebral bodies. Even when permanent paralysis is anticipated, such restoration is desirable, in order to relieve pain, to prevent skin pressure over kyphotic protrusions and to allow whatever weight-bearing may be possible.

In the cervical region, some orthopedic surgeons employ manipulative reduction, but this method is not without danger and, in most cases, the same results can be obtained by the maintenance of strong traction. For the latter purpose, the skeletal traction tongs of Crutchfield offer by far the most satisfactory method. This device allows the application of strong traction without the interference with jaw movements which is inherent in all halter methods.

In lower portions of the spine, on the other hand, manipulative treatment accomplishes excellent, often brilliant, results. Potential danger to the cord may be minimized, if necessary, by preliminary laminectomy.

It has long been customary to treat such cases by hyperextension with or without application of a certain amount of traction. Dr. Eugene Regen, of our Orthopedic Department, has added the use of very strong traction (up to 275 lbs. equivalent). The hyperextended position is achieved and a plaster jacket applied while this traction is being maintained. Well-nigh miraculous reduction of crushed vertebral bodies and extreme dislocations may be obtained in this way.

Care of the skin. In no other field of medicine is constant, painstaking nursing care so important. Skin which is deprived of its nerve supply, and which doubtless has its blood supply impaired simply fades away under even slight pressure. Huge decubitus ulcers may develop in an astoundingly short time over the bony prominences—the sacrum, greater trochanters, heels and malleoli.

There is no short cut to the prevention of bed-sores. Frequent changes of position, gentle massage of "pressure-points," use of rubber rings, "doughnuts," air or sponge-rubber mattresses and maintenance of a constantly dry bed and skin are the prime essentials. Once ulceration has developed, it must never be subjected to pressure and must be handled with all the aseptic care accorded to a major surgical wound. Necrotic tissue should be cut away, a bland ointment applied to the skin edges and the dressing changed frequently. A minimal amount of adhesive tape should be used. Oral sulfonamide therapy will help to prevent spreading infection here as in the urinary system.

Care of the bladder and bowel. Death in patients who survive the immediate posttraumatic period after cord injury is usually the direct or indirect result of urinary infection. Retention of urine is the most frequent disorder, but incontinence may also be present.

While the patient is in the hospital, undoubtedly the best solution is some form of tidal drainage. This device, first suggested by Munro, has been modified by many surgeons, but all maintain its basic principle of automatic irrigation, intermittent drainage and maintenance of a satisfactory bladder capacity.

Later, there are two alternatives: the patient may be allowed to develop an "automatic" bladder, which overflows when a certain degree of distention is reached; or a suprapubic cystostomy may be done. The choice depends upon two factors.

First, if any degree of recovery of voluntary bladder function may be expected, the automatic bladder is the lesser of two evils, for it at least prevents contracture of the bladder into a small, fibrous sac with no elasticity or future usefulness as a reservoir. If the normal function will never return, this consideration is negligible.

Second, the economic and domestic status of the patient is important. In poor surroundings, with little opportunity for adequate nursing care and little likelihood of frequent urinalysis and observation by the physician, the suprapubic drainage offers less likelihood of ascending urinary infection than the constant retention of the automatic bladder. One advantage of this method is that the suprapubic sinus may be allowed to close at a later time if it seems best. When incontinence is present, cystostomy is essential.

In a few favorable instances, the patient can maintain a tidal drainage system at home.

Treatment of Penetrating Wounds

The presence of an external wound introduces the problem of infection and hence alters the necessary therapy. Every wound which may involve the spinal canal is a potential source of meningitis and is therefore an acute surgical emergency. Such wounds should be treated like those elsewhere in their superficial portions—careful cleansing, debridement, and removal of foreign bodies.

However, the decision as to whether laminectomy and opening of the dura are to be carried out must depend upon two factors: whether there is blood in the cerebrospinal fluid and whether foreign bodies are demonstrable within the spinal canal. The dura should *never* be opened unless the spinal fluid contains blood. Foreign

bodies should be removed only if accessible or if they impinge upon the lumen of the canal.

If the wound is seen early and frank infection is not present, the dura (if opened) should be closed and the wound should not be drained. Sulfanomide therapy will greatly reduce the likelihood of infection, but drugs should *never* be placed in direct contact with the spinal cord.

The problems of cord injury, per se, and of skin and bladder are the same as in closed injuries.

Late Complications

Aside from the difficulties with skin and bladder already discussed, two problems may arise: the one is inadequate stability of the spine for bearing weight and the other is pain, due to arachnoiditis or nerve root compression.

Instability of the spine is best treated by the orthopedist with some form of spinal fusion operation, which gives eminently satisfactory results.

The problem of nerve root pain should be attacked surgically, for the only alternative is drug addiction. In a few cases, section of specific nerve roots will be feasible, but usually chordotomy should be performed. This procedure consists of section of the antero-lateral spinothalamic tracts at a level well above the injury. These tracts transmit all somatic painful sensation and their division permanently abolishes perception of pain below the level of section.

Conclusion

It may be seen that the picture is by no means bright. Destruction of structures in the spinal cord, once accomplished, is irreparable. Nevertheless, it remains that the surgeon, since he must deal with these cases, has need of accurate knowledge, meticulous judgment and sympathetic patience in properly handling the problems presented. And it is seen, too, that he may sometimes accomplish much when little is expected.

STERILIZATION OF THE SULFONAMIDES FOR LOCAL USE

A Neglected Problem

DOUGLAS LINDSEY, B. S.*

New Orleans

THE sterilization of the sulfonamides for use in the abdominal cavity and in clean wounds is a problem; not because it presents technical difficulties, but because of combined indifference and misunderstanding on the part of surgeons and operating room nurses.

The local prophylactic use of sulfonamide powders in clean wounds or in body cavities at operation has recently become very popular. Several authors offer good evidence that the popularity of this procedure has caused it to be used in excess of sound indications.[1,10] Many surgeons have quickly approved of the benefits of the technic but have not paid sufficient attention to its disadvantages or to the reservations with which it was originally recommended.

Certain complications of the procedure, such as tissue damage and interference with wound healing, have been discussed by other authors.[1,10] It is not my purpose, nor am I qualified, to pass judgment on this aspect of the problem. However, it has come to my attention that a practical point in the local use of the sulfonamides has been neglected: that, even when the indications for the procedure are unquestionable, there is frequently a serious error in technic.

A recent personal survey of the private hospitals of New Orleans reveals that *half* of them do not supply in sterile form the sulfonamides that are used locally for the prophylaxis of infection. My understanding of the factors responsible for this condition leads me to believe it is not at all confined to New Orleans. A survey of the recent literature confirms this impression.

The attitude of the individual surgeon toward the sterility of the sulfonamide which he is placing on or in his patient's yet uninfected tissues puts him in one of two groups: those whose surgical conscience or judgment dictates that they use only sterile preparations; and those who realize that the compounds they are using are not sterile, but who are not concerned about it.

To the surgeons of the first group the author has presented all the data necessary: the fact that the sulfonamides are not universally supplied sterile when they are to be used in a clean field.

*Fourth year student, Louisiana State University School of Medicine; and Extern, the French Hospital.

This will surprise many of them, as I have already found out by personally questioning a few members of this group. The realization of the situation will enable them to settle the problem themselves by specifically requesting sterile preparations.

The surgeons of the second group might do well to consider seriously whether they are not allowing the popular reputation of a drug to obscure the importance of a fundamental surgical principle. They should review the literature that introduced the procedure of prophylactic implantation of the sulfonamides in clean wounds.[5, 6, 8] In these and similar articles they will find one or two short sentences that they missed the first time: ". . . The compound used was sterilized by autoclaving . . . "

The general attitude of the surgeons of this latter group is similar to that expressed by an operating room technician at one of the hospitals where these compounds are not supplied sterile: "If sulfanilamide does all the things they say it will it ought to sterilize itself." Truly the powers of the sulfonamides are little short of miraculous. They have revolutionized the treatment of many medical and surgical conditions. But they should not be allowed to overrule the cardinal principle of asepsis. It does not fit any system of logic to put an unsterile substance on a sterile towel and pass it into a sterile field.

One of the properties that gave the sulfonamides such a hallowed reputation in the eyes of the public when they were first introduced is the great difference in their powers *in vitro* and *in vivo*. That should not be forgotten now. The accomplishments of the sulfonamides in various infections within the body cannot be carried over to include insurance against contamination within a glass jar.

It is true that the local bacteriostatic effect of the sulfonamides would take care of all or most of the contaminants introduced along with them into a clean field. However, several factors make total dependence on this bacteriostatic action uncertain and dangerous. The contaminants might include spores capable of living in high concentration of the sulfonamide for a long time; the possibility of tetanus is remote, but certainly not absent.* The "lag period" of several hours before the sulfonamide begins to act might allow a contaminant to establish itself out of reach of the sulfonamide. The local concentration of the sulfonamide in certain portions of the wound or peritoneal cavity might be low enough to allow the organism to keep alive even after the lag period is over; not even 200 mg. per cent in a good medium

*As this paper was being completed, a brief note has appeared, describing a case of fatal tetanus after intraperitoneal implantation of sulfapyridine. The infection was traced to contamination of the container.[11]

will prevent the growth of certain organisms.[3] Even sterile sulfonamides are capable of causing tissue damage and necrosis;[10] damaged areas would offer good fields for growth of surviving organisms. The local bacteriostatic effect of the sulfonamides is inversely proportional to the number of bacteria present; gross contamination cannot be taken care of.

Of course it will be argued that gross contamination is not likely to occur in a chemically pure crystalline compound handled with ordinary clean technic. But when the large stock bottle of the sulfonamide is repeatedly opened to remove small amounts for immediate use, and when these transfers are carried out with the idea that the compound is self-sterilizing, the possibility of significant contamination of the stock bottle cannot be ignored. The author has cultured a variety of organisms from a few milligrams of sulfonamide powder removed from a stock bottle opened relatively few times.

The argument that no complications have been reported traced to contamination of a sulfonamide does not justify the use of the unsterile compound in a clean field. The recuperative powers of the human body are marvelous, but not infallible. I have seen a patient receive an intravenous injection of plain tap water with absolutely no ill effects, but no surgeon would approve such a procedure. Even if such complications did occur they could be easily laid to something else; the reputation of the sulfonamides would prevent their indictment.

A factor that has played a great part in supporting the use of unsterile sulfonamides is the common misconception that these compounds cannot be sterilized without decomposition. It is true that they are broken down by high temperatures, but they may be safely sterilized by dry heat at 120° C. for 20 minutes or by autoclaving at 15 pounds pressure for 20 minutes. The latter technic will cause some caking of the powdered compound but it can be repulverized.

BIBLIOGRAPHY

1. Bick, E. M.: Observations on the Topical Use of Sulfanomide Derivatives, J. A. M. A. *118:* 511-513 (Feb. 14) 1942.
2. Hagna, L. W., and McBee, P.: Local and Parenteral Use of Sulfonamides, South. Med. & Surg. *104:* 16-17 (Jan.) 1942.
3. Heath, F. K., and Province, W. D.: The Preservation of Human Plasma; Report of Studies with the Sulfonamide Compounds, J. A. M. A. *118:* 1034-1037 (March 28) 1942.
4. Jervey, W. St. J.: The Local Use of Sulfonamides, South. Med. & Surg. *103:* 643-645 (Dec.) 1941.
5. Key, J. A.: The Use of Sulfanilamide and Sulfathiazole in Orthopedic Surgery, J. A. M. A., *117:* 409-412 (Aug. 9) 1941.

6. Key, J. A., and Burford, T. H.: The Prophylactic Implantation of Sulfanilamide in Clean Operative Wounds for the Reduction of Postoperative Infections, Surg., Gynec. & Obst. *73:* 324-332 (Sept.) 1941.
7. Key, J. A., and Frankel, C. J.: The Local Use of Sulfanilamide, Sulfapyridine, and Sulfamethylthiazol, Ann. Surg. *113:* 284-297 (Feb.) 1941.
8. Key, J. A.; Frankel, C. J., and Burford, T. H.: The Local Use of Sulfanilamide in Various Tissues, J. Bone & Joint Surg. *22:* 952-958 (Oct.) 1940.
9. Lee, Q. B.: The Use of Sulfanilamide Powder in Abdominal Surgery, South. Surgeon *10*: 680-685 (Sept.) 1941.
10. Taylor, F. W.: The Misuse of Sulfonamide Compounds, J. A. M. A. *118:* 959-961 (March 21) 1942.
11. Tetanus After Local Chemotherapy, Lancet *1:* 770 (June 27) 1942.

MASSIVE GASTROINTESTINAL HEMORRHAGE CONCOMITANT WITH CHOLECYSTITIS

E. G. Laird, M. D., F. A. C. S.
A. M. Gehret, M. D.
and
L. J. Rigney, M. D.
Wilmington, Delaware

MASSIVE gastrointestinal hemorrhage resulting from or concomitant with chronic gallbladder disease is an uncommon and comparatively unrecognized syndrome. An adequate number of such cases have been reported, but they have not been stressed enough to cause this relationship to be generally recognized. In his textbook, Stevens[1] says that "hematemesis is an occasional accompaniment of cholecystitis"; but he fails to mention chronic cholecystitis when he lists the common causes of enterorrhagia.

Recently Dr. Laird encountered a massive and nearly fatal postoperative gastrointestinal hemorrhage in a patient with severe gallbladder pathology and coincidentally learned that a second case, almost identical with it, was at that time under the care of Dr. Gehret. The two cases presented points of contrast which caused us to review the literature on this subject and report these two cases.

We have endeavored to carry out more exhaustive investigations than have been done on previous cases, such as prothrombin determinations, gastrascopy, esophagoscopy, etc.

Our contributions to the discovery of the essential cause of the hemorrhage are minimal, as were also those of the previous authors; but we are able to show certain facts in relation to the prognosis and treatment of this condition and that more intensive thought and study should be devoted to it in the future, especially in the light of our recent knowledge concerning the role of the liver in hemorrhagic syndromes.

The most complete work on this subject which has come to our attention is by White and Jankelson[2] at the Boston City Hospital. They gave a comprehensive review of the 12 cases occurring in the medical literature up to 1931 and added 6 of their own. Judd, Marshall, and Hartwell[3] of the Mayo Clinic, reported another instance, a physician, in 1930, while Seligman[4] in St. Louis added still another as recently as 1936.

The following two cases were personally observed by the authors:

Case Reports

Case 1. Mrs. M. D., aged 53, was first admitted to the Wilmington General Hospital on Jan. 12, 1941, giving a history of a spell of weakness, clammy

skin and shortness of breath one year previously. Since then she had suffered from shortness of breath, paroxysmal dyspnea, occasional unexplained vomiting, bloody stools and dark brown urine. She had had six children and passed through a normal menopause at the age of forty. She had never been jaundiced. Examination revealed a definite anemia, B.P. 142/68, a soft apical systolic murmur, the liver extending four fingerbreadths below the rib margin. There was slight pitting edema of the extremities. The electrocardiogram was interpreted as negative. X-rays of the stomach and intestines revealed only hypermotility of the pylorus. Laboratory studies: Red blood cells 2,300,000; hemoglobin 30 per cent; platelets 332,000; white blood cells 14,800; polymorphonuclears 86 per cent; monocytes 14 per cent; blood urea nitrogen 11 mg. per cent; blood sugar 166 mg. per cent; urine not remarkable; stools positive (4 plus) for occult blood. Bleeding time 1 minute; coagulation time 3 minutes.

On February 6 she was discharged with a diagnosis of mitral and aortic heart disease and secondary anemia.

Second admission was six months later. Her complaint at this time was of weakness. After thirteen days of observation she was discharged under the diagnosis of "probable bleeding peptic ulcer." She had received two blood transfusions.

In July, 1941, a complete gastrointestinal series by Dr. Rigney revealed the suggestion of a defect in the duodenal cap. This was interpreted as an ulcer. She was readmitted on Oct. 4, 1941, presenting essentially the same picture as before. On November 8, Dr. Gehret explored the abdomen and found a normal intestinal tract except for adhesions between the duodenum and the gallbladder. The latter was distended, thickened, and contained several large stones in the pouch of Hartman. On opening the fundus, it was found that the wall had eroded into the substance of the liver. A simple cholecystostomy was performed after removing the stones. On November 13, she passed tarry stools. She was given repeated transfusions. Laboratory tests reported at this time: Red blood cells 2,000,000; hemoglobin 30 per cent; prothrombin time on November 13 (before receiving any vitamin K) 80 per cent normal; on November 24, normal; gastric analysis showed no free hydrochloric acid in any specimen and a maximum total hydrochloric acid of 5; stools intermittently positive for occult blood.

On December 6, she was discharged finally to the Jefferson Hospital, Philadelphia, for further study. A complete gastrointestinal roentgenologic study, esophagoscopy, and gastroscopy were negative. Hematologic investigation revealed "a moderate anemia, hypochromic in type, slight thrombopenia and prolongation of the bleeding time. Plasma prothrombin and ascorbic acid blood levels were normal." The exact nature of the melena was not ascertained, but was attributed to the patient's vascular heart disease.

On April 28, 1942, she was readmitted with the same complaints. Hemoglobin 38 per cent; red blood cells 2,900,000; white blood cells 3,300. She received four transfusions, and her blood count was brought up to hemoglobin 79 per cent; red blood cells 4,170,000; white blood cells 4,000.

At operation exploration of the abdomen was essentially negative except for numerous adhesions at the site of the previous operation. The fundus of the gallbladder was markedly thickened and sclerosed as a result of the previous cholecystostomy. A cholecystectomy was done.

The patient had an uneventful convalescence and was discharged on June 2, two weeks after operation. On the day previous to discharge, the blood count was: hemoglobin 78 per cent; red blood cells 4,110,000; white blood cells 6,650.

There is one point which might be mentioned: namely, that following cholecystectomy, the leukopenia which the patient had presented was improved. Since operation and to the present date, there have been no recurrences of the gastrointestinal hemorrhages, although this period of observation has, of necessity, been comparatively short.

Case 2. M. M., female, aged 57, was admitted to the Wilmington General Hospital for chronic cholecystitis with cholelithiasis. Twenty years previously she had had an ovarian cyst removed and had not menstruated since then. Eighteen months after this operation, following an attack of upper abdominal pain radiating to the right shoulder blade, cholecystotomy and appendectomy were performed. Stones were found in the gallbladder. She had remained well until 12 years previous to this admission, when the gallbladder attacks had recurred. Following a severe attack eight days before with nausea and vomiting, she was admitted for operation. There was no jaundice nor abnormal stool. Laboratory studies: Red blood cells 3,000,000; hemoglobin 60 per cent; white blood cells 8,600; polymorphonuclears 60 per cent; blood urea nitrogen 12 mg. per cent; blood sugar 107 mg. per cent; icterus index 3, urine not remarkable; stools were negative to the benzidine test for occult blood and for ova, parasites, etc., but were somewhat light of color; gastric analysis showed a fasting free hydrochloric of 11 and a total hydrochloric of 13. After histamine (.5 mgm.) it rose to 40 and 60 degrees respectively. Gallbladder x-rays made 3 months previously by Dr. Rigney revealed "either calcification of the gallbladder or two large stones." The physical examination was not remarkable except for obesity, a moderate pallor of the mucous membranes, and a third degree perineal tear. Blood pressure was 140/85. The abdomen showed two healed surgical scars and a liver enlarged two fingerbreadths below the costal margin and extending over the whole epigastrium to the left hypochondrium. Tenderness was present only in the gallbladder region.

On October 22 an exploration was undertaken. Frozen in dense adhesions, a large distended gallbladder containing several stones was found. The liver edge was somewhat blunted but there was no marked cirrhosis. The stomach and duodenum were entirely normal except for adhesions distorting the latter. In removing the gallbladder it was found that the posterior wall had eroded, causing an abscess into the liver substance.

The patient developed a postoperative, bilateral bronchopneumonia from which she recovered satisfactorily. On the eighth day after operation at 12:30 a.m. she suddenly vomited a large amount of dark clotted and unclotted blood. She was given vitamin K (1.6 mg.) subcutaneously and repeated doses of Amphogel. Twelve hours later the prothrombin time was 90 per cent of the normal, with a 21 per cent hematocrit. Blood serum specific gravity was 1.016 and serum proteins 3.027. She responded to multiple transfusions. On November 13 the stools were negative to the benzidine test for occult blood. She was discharged November 15 in good condition. She has been essentially symptom-free to date on an average low-fat diet, without signs of recurrent hemorrhage.

In an effort to locate the source of the bleeding, Dr. Rigney repeated the gastrointestinal series on Feb. 16, 1942, and studied the esophagus under the fluoroscope with a thick barium mixture, finding only phrenospasm, prom-

inence of the gastric rugae without defects in the outline of the stomach or duodenum; slight dilatation of the duodenum but no stasis. Esophagoscopy and gastroscopy were arranged, but the patient refused to submit to them.

Whereas these two cases might have been even more thoroughly studied under other circumstances, we feel that they have probably been investigated more completely than any previously reported and may be definitely informative in a negative way.

The following facts concerning these two cases are worthy of note:

1. In one instance the plasma prothrombin was found to be essentially normal while the patient was still bleeding. This was before the administration of vitamin K. In the other, the prothrombin was 100 per cent normal within 12 hours after the injection of vitamin K. To our knowledge, these are the first two cases in which the prothrombin time was recorded, and it would not appear to us that the lack of production of prothrombin is an essential factor in these hemorrhages. Numerous authors, including Judd[5] and Graham[6], have observed that there is a varying degree of hepatitis with almost every cholecystitis. Since the production of prothrombin takes place in the liver, one must assume that the degree of liver damage in these hemorrhagic cases is not such that this particular function is inhibited to a great extent.

2. The bleeding and coagulation time were tested only in Case 1 and were within normal limits.

3. X-rays of the gastrointestinal tract have proven to be of little positive aid. The most common finding in the literature has been a moderate deformity of the first portion of the duodenum, sometimes suspected to be an ulcer, sometimes hyperperistalsis or spasm. In no instance, however, has any suggestion of an ulcer been found at operation, although the stomach and duodenum have been opened for direct inspection in several instances.

It is interesting that both of our cases happened to be studied roentgenologically by Dr. Rigney both before and after operation. The only notable finding was a patent, relaxed ampulla postoperatively in Case 2. This has been described by Judd[5] and others.

In Case 2 a phrenospasm was noted at the postoperative esophagoscopy.

4. Gastroscopy and esophagoscopy were permitted in only one instance (Case 1). No abnormalities were found. This patient also presented the clinical picture of valvular heart disease, but the electrocardiogram was essentially normal. The cardiovascular system of the other patient was without abnormality. Blood pressures were 140/86 and 142/68 respectively in these two cases. Cardio-

vascular diseases has not been striking in the cases previously reported.

5. A complete study of the fluid balance and hematology was obtained in only one of our cases (Case 2). On the morning following the hemorrhage the following laboratory tests were made: Hemoglobin 50 per cent; red blood cells 2,600,000; white blood cells 10,400; hematocrit 21 per cent; serum specific gravity 1.016; serum proteins 3.27 mg. per cent.

The interpretation of these findings is rather difficult because, whereas they would appear to show severe hemorrhage and an excessive fluid intake, the patient had been receiving only approximately 1500 c.c. of fluids per day prior to the hemorrhage and was at no time edematous. No intravenous fluids had been administered prior to the determinations and the urine output ranged from 500 c.c. to 600 c.c. daily. It would appear, therefore, that the laboratory tests were inconclusive.

6. In both cases it was found at operation that the posterior wall of the gallbladder had eroded and that the infection had direct contact with the liver parenchyma.

This certainly would increase the toxic damage to the liver cells, and we feel that this fact is worthy of note as *one* of the probable causes of the hemorrhage, which is presumably the result of a deficient liver function, the exact nature of which we are unable to suggest.

7. In spite of the apparent liver damage, neither case was icteric, and this was likewise true in most of those cases reported by other authors.

8. The renal function was inadequately determined in both patients, but, other than a transient, slight albuminuria, the kidneys were normal as determined by the specific gravity of the urine and by urea excretion.

9. The last and most striking fact to be brought out—and one which has been consistently substantiated by the findings of others—is that, when a simple cholecystostomy has been done and the diseased gallbladder has been left in the abdomen, the hemorrhages have recurred; whereas if the gallbladder has been removed, the hemorrhages have ceased. Some of these cases have been followed for over 5 years.

Conclusions

1. Twenty cases of massive, gastrointestinal hemorrhage concomitant with gallbladder disease have been reported in the literature.

2. We have submitted two additional similar cases which we have attempted to study more completely than has been done previously.

3. The source of and cause for these hemorrhages has never been ascertained.

4. Several important facts concerning this condition have been emphasized, the most striking being that ablation of the diseased gallbladder produces a cessation of the hemorrhages, which almost inevitably recur if this organ is allowed to remain in the body.

5. The two cases submitted present unusually interesting points of comparison and contrast.

6. This syndrome is not generally recognized and deserves more study.

BIBLIOGRAPHY

1. Stevens, A. A.: The Practice of Medicine, Philadelphia, W. B. Saunders, 1926, p. 544.
2. White, F. W., and Jankelson, I. R.: Gastro-Intestinal Hemorrhage in the Disease of the Gallbladder, New England J. Med. *205:*793, 1931.
3. Judd, E. S., Marshall, J. M., and Hartwell, S. W.: S. Clin. North America, *10:*10, 1930.
4. Seligman, E.: A Case of Hematemesis Following Cholecystectomy, Weekly Bull. St. Louis Med. Soc. *30:*353, 1935-1936.
5. Judd, E. S.: Relation of the Liver and the Pancreas to Infection of the Gallbladder, J.A.M.A. *77:*197, 1921.

DIAGNOSTIC CURETTAGE

ROBERT B. GREENBLATT, M. D.

Augusta, Ga.

A time-honored therapeutic and diagnostic measure has been and is the surgical procedure known as dilatation and curettage. Its chief use heretofore has been in the therapy of menometrorrhagia, dysmenorrhea and incomplete abortion. Its use for diagnostic purposes in general has been limited to suspected uterine malignancy. With the advent of clearer concepts in gynecologic endocrinology frequent resort to the study of the endometrium is necessary. To hospitalize a patient for a surgical dilatation and curettage in such instances is not only unnecessary but also an added and expensive burden. Today, suction curettage is readily undertaken as an office procedure. Medical curettage may be performed simply through hormonal administration. Both procedures are diagnostic measures of much import to the gynecologist.

Suction Curettage or Endometrial Biopsy

Many types of curets are in use; either the Randall curet which has a smooth fenestrum, or the Novak curet which has a serrated fenestrum, is generally employed. Examine the uterus for size and position and rule out pregnancy. Use a speculum, small or large, clean the cervix with zephiran or merthiolate. Without touching the vaginal wall insert the sterile curet and pass it through the cervix into the uterus (fig. 1). The position of the curet depends on the position of the uterus, that is, whether it is anteflexed or retroverted (fig. 2). Use a 10 c.c. sterile syringe with a metal adaptor or Luer-Lok. Obtain the endometrium by suction while gently scraping the surface of the endometrial cavity and then withdrawing the curet. Wash out the curettings with normal saline. The blood and mucous may thus be distinguished from the purplish pink curettings. The latter sink to the bottom and these are then placed in 10 per cent formaldehyde solution and sent to the pathologist for histologic study. If glycogen stains are desired the tissue should not be washed out with saline but placed immediately in a solution of 5 per cent formaldehyde and 95 per cent alcohol.

From the Department of Experimental Medicine, University of Georgia School of Medicine.

Abridged from an address delivered Sept. 17, 1942 before the Tenth District Medical Society of Georgia and the Georgia Section of the Southeastern Surgical Congress at Athens, Georgia.

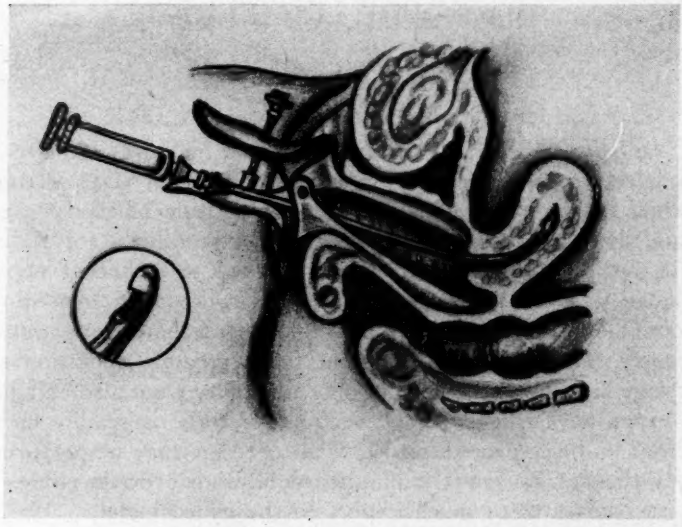

Fig. 1. Randall curet (smooth fenestrum) in position in uterine cavity.

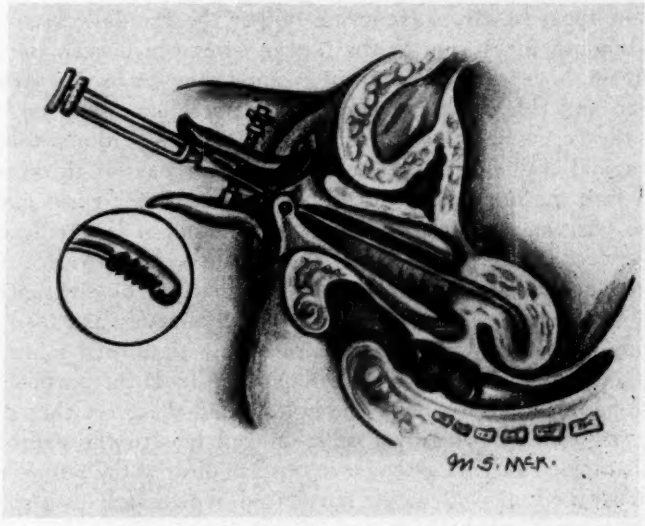

Fig. 2. Novak curet (serrated fenestrum) in uterus in retroverted position.

When to Perform Suction Curettage

In *amenorrhea,* perform the suction curettage at least 30 to 40 days after the last menstrual period.

In *menorrhagia,* the suction curettage may be performed at any time, preferably however as soon as possible after the start of a phase of bleeding.

In *dysmenorrhea,* perform the suction curettage several days before the onset of menses.

Fig. 3. Atrophic or hypoestrogenic endometrium.

In *sterility,* suction curettage should be performed soon after the onset of menses to avoid disturbing a possible pregnancy.

The study of the endometrium is informative for it not only mirrors ovarian activity, but also yields some clue as to follicle stimulating hormone secretion of the anterior pituitary gland. For instance, if the endometrium obtained is atrophic (fig. 3), it indicates that there is an estrogen deficit which may be due to primary ovarian failure (look for other stigmata of excess follicle stimulating hormone secretion) or may be due to hypopituitarism (lack of follicle stimulating hormone) with secondary ovarian failure.

A persistent estrogenic endometrium obtained on the 25th day of a 28 to 30 day cycle indicates that ovulation has not taken place, and that either the proliferative phase or a persistent resting en-

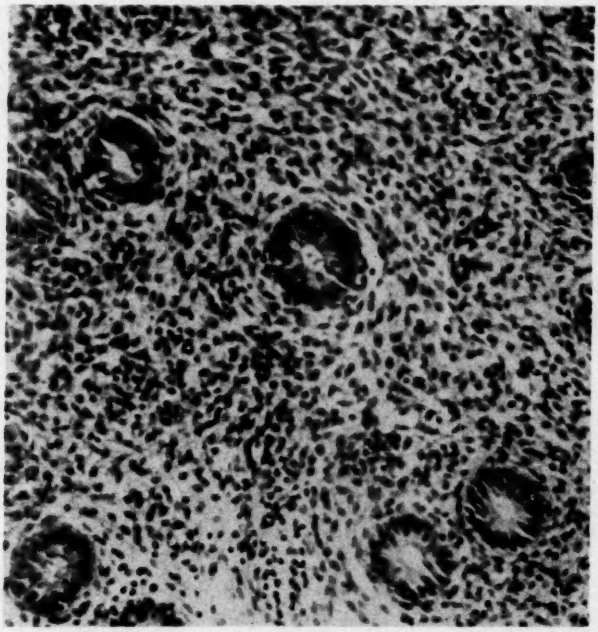

Fig. 4. Cystic glandular hyperplasia of endometrium.

dometrium have accordingly continued. A cystic glandular hyperplasia (fig. 4) indicates that there is marked local response to an excess of endogenous or exogenous estrogens, or it may be the indirect result of disturbances in the metabolism of normal quantities of estrogen, or it may be due to continuous stimulation of subnormal quantities of estrogens over a long period of time as in hypogonadal metropathia hemorrhagica. An imperfect progestinal or mixed type of endometrium (fig. 5) may signify faulty corpus luteum production. A true secretory endometrium is proof of the occurrence of ovulation and corpus luteum formation.

Medical Curettage

Medical curettage is the term coined by Albright[1] to denote the induction of endometrial shedding by hormonal means and is performed for two purposes:

1. It may be employed to ascertain whether amenorrhea is due to complete estrogen deficit and secondary ovarian failure or

whether it is due to endocrine imbalance without estrogen depletion. The parenteral administration of 10 mg. of progesterone daily for five days will be followed within seventy-two hours by a menstrual

Fig. 5. Imperfect progestinal or mixed type of endometrium.

flow if estrogens are present. Failure will result in the absence of estrogen. A menstrual flow may be obtained in complete estrogen deficit states by first administering a course of estrogens which is then followed by a short course of progesterone (5 mg. daily for 3 to 5 days). The very fact that estrogens are necessary before progesterone withdrawal bleeding may be induced is indicative of an estrogen lack.

2. Medical curettage may be employed as a therapeutic procedure in the amenorrhea-menorrhagia syndrome of metropathia hemorrhagica to induce shedding of the thickened hyperplastic endometrium.

REFERENCE

1. Albright, F.: Metropathia Hemorrhagica, J. Maine M. A. *29:* 235-238 (Nov.) 1938

LOBECTOMY FOR SUPPURATIVE DISEASE OF THE LUNG

ROLLIN A. DANIEL, JR., M. D.
Nashville

EXCISION of lung for suppurative disease has been performed in the Vanderbilt University Hospital in 22 cases. Eight of these patients were operated upon prior to the year 1935, by the method formerly employed by Graham and known as "cautery pneumectomy." This operation consists of the burning away of portions of the lung in stages, the wound being kept open by means of gauze packs between each stage. The present condition of all but two of these patients is known. Five patients are dead, one dying on the third postoperative day of pneumonia, all the others within four months after the last stage was completed. Two patients had repeated severe secondary hemorrhages following operation but neither died of this complication. One patient is alive and well 12 years after operation for multiple abscesses. Two patients were in good condition when last seen, several months after operation.

These operations were performed for lung abscess (multiple) in four cases, for bronchiectasis in three and for metastatic sarcoma and lung abscess in one. The one patient known to be well had a large abscess. The three patients who had bronchiectasis all died.

Since 1936 resection of lung for suppurative disease has been performed upon 14 patients. One patient had total left pneumonectomy because of a mistaken diagnosis, a large abscess in the upper lobe being thought to be a neoplasm. This patient is living. Lobectomy was performed in the remaining 13 cases. There were two immediate operative deaths among this group of patients. Two other patients died two and one-half and three months after operation, one of a septic embolus to the brain and the other of a persistent bronchial fistula. This is a total operative mortality rate of 28.6 per cent. Ten (71.4 per cent) are living and all of these are improved or are completely well. Two of them have persistent bronchial fistulae. Only the last seven patients were operated upon by the author, and these during the last year. These will be considered in some detail.

The treatment of suppurative disease of the lung has always been a difficult problem. The mortality rate of untreated cases is extremely high. About 25 per cent of patients with lung abscess can be cured by conservative measures alone, the mortality rate among these patients being about 30 per cent. The remainder re-

From the Department of Surgery, Vanderbilt University School of Medicine. Read before the Nashville Surgical Society, October 9, 1942.

main essentially the same over a period of years. The mortality rate in bronchiectasis is difficult to determine accurately, since there are few recorded studies in which groups of patients with the disease have been followed carefully for long periods of time. However, Bradshaw, Putney and Clerf[1] recently reported on 171 patients with bronchiectasis. The diagnosis of all these patients was established between the years 1925 and 1935. Of these patients 34.5 per cent died from bronchiectasis or its complications. Perry and King[2] were able to follow 211 similar patients and found a mortality rate of 31 per cent. Riggins[3] and Ogilvie[4] report somewhat lower mortality rates but the former stresses the importance of a consideration of the morbidity which accompanies the disease. Churchill[5] has stressed also the "psychologic" aspects of the disease and believes that in the majority of cases lobectomy is desirable on this basis alone. The relative number of social and economic "misfits" is enormous among this group of patients. The onset of the disease occurs most frequently during childhood. Physical activity, important in the mental development of the child, is often greatly restricted. Because of the fetid breath and paroxysms of cough productive of foul sputum, social contacts are difficult or impossible and eventually are shunned by the patient himself. These same factors often make regular employment impossible and either because of their illness or because of discouragement many patients become economic liabilities. Suicides occur occasionally for no other apparent reason than the presence of the disease itself.[3]

It is certainly true that all cases of bronchiectasis are not progressive and that some patients die in old age, of other causes, after a useful and almost normal life. Such instances are unusual, however, and ultimate prognosis in any given case is unpredictable.

We know, then, that lung abscess, either single or multiple, and bronchiectasis are fatal diseases. Moreover the morbidity which accompanies a large proportion of conservatively treated or untreated patients is in itself an indication for radical therapy. The causes of death are, most commonly, pneumonia, pulmonary gangrene and empyema, hemorrhage and brain abscess.

The surgical treatment of these diseases has been almost as unsatisfactory as has conservative therapy until recently. During the last ten years, however, notable advances have been made and the relative number of patients who are either cured or greatly improved is increasing. In 1932 Neuhof[6] reported his experiences with putrid abscess of the lung, and emphasized the need for early drainage of lung abscess. He found that by properly localizing the abscess drainage could usually be accomplished in one stage. He later reported a mortality rate of 3.6 per cent and stated, "Putrid

lung abscess is potentially a surgical lesion from the outset. There is no proved method of definitely influencing the course than by open operation." We agree in principle with Neuhof's ideas, and although our experience with early one stage drainage has been better than in cases treated conservatively or by delayed operation, our results have not been as good and recurrence of abscess has occurred more frequently than in his experience. Shaw[7] has recently published his results in the use of early drainage as advocated by Neuhof and it is of importance to note that whereas only 5 per cent of patients with a single abscess died, 30 per cent of patients with multiple abscesses died and only 23.1 per cent were well at the time of publication of his paper. These results are comparable with those generally obtained with conservative treatment or with none at all. Pulmonary resection has not been generally advocated as a method of treatment of lung abscess, but we believe that, as greater experience is gained, eradication of the disease by this means will be resorted to more frequently. Many abscesses are located near the periphery of the lung and adhesions existing between the wall of the abscesses and the chest wall make the mobilization of the diseased lung hazardous. Excision of the lung is therefore more difficult than with uncomplicated bronchiectasis or with neoplasms.

The most satisfactory results from pneumonectomy have been achieved during the last several years in the treatment of uncomplicated bronchiectasis. In 1927 Archibald wrote that the problem of lobectomy is chiefly concerned with the question of operative mortality and that it will be solved when the mortality has been reduced to 20 per cent or even less. In 1933 Alexander collected 115 cases from eight leading American and foreign clinics and found the operative mortality rate to be 21.5 per cent. These operations had been performed by many variable methods, including the cautery pneumectomy of Graham and the use of the cord snare tourniquet of Shenstone and Janes which is used by many surgeons today. The use of pressure packs combined with the ligation of the hilar structures and partial exteriorization of the lobe with subsequent gangrene and sloughing away of the lung was resorted to by Sauerbruch and others, and Lilienthal advocated the use of a bag made of rubber dam to isolate the diseased lobe. Alexander strongly advocated the two-stage operation, emphasizing the importance of adhesions between the undiseased lung and parietal pleura in the control of infection.

The general operative mortality rate during the last few years has fallen to less than 20 per cent. Churchill, who has probably had a greater experience in the surgical treatment of bronchiectasis

than anyone else, reported, in 1940, the remarkably low mortality rate of 2.4 per cent in 124 lobectomies for bronchiectasis.[10]

The technic of one stage total lobectomy will not be discussed in detail. Meticulous dissection of the hilar structures and the avoidance of tissue injury and mass ligation of tissues are probably the most important considerations as regards the avoidance of infection and its complications.

Report of Cases

During the last year seven lobectomies have been performed in the Vanderbilt Hospital for suppurative disease. There were two hospital deaths, one of which occurred in the case of a 31 year old white man who had multiple abscesses in the right lower lobe. Death occurred five days after operation. Another patient had a bronchopleural fistula when he died three months after operation from malnutrition. This patient was a 57 year old white man with right lower lobe bronchiectasis who was operated upon as an emergency, after having had repeated massive hemorrhages over a period of 10 days. Five patients are alive and in good health at this time. A brief summary of these cases follows (See Table I).

Case 1. W. D. Y., a 12 year old white male, admitted Aug. 12, 1941, with a history of chronic cough productive of foul sputum for six years, following whooping cough. During the last eight months cough had been worst and for five months he had had purulent drainage from the left ear and had been known to have chronic maxillary sinusitis. For a year he had carried out faithfully a regime of postural drainage without apparent improvement. Also during the last year his activity had been markedly limited because of shortness of breath on exertion.

Examination revealed a well developed white boy who was rather pale and appeared to be chronically ill. Numerous coarse and fine sticky rales could be heard over the left base posteriorly. Bronchograms revealed dilatation and clubbing of the bronchi in the left lower lobe and the lingula segment of the left upper lobe.

Left lower lobectomy was performed on August 14. He developed a small empyema which was drained three and one-half weeks later. His convalescence was otherwise uneventful and he has been greatly improved since then. On July 12, 1942, he re-entered the hospital for operation on his maxillary sinuses.

The lingula segment of the upper lobe was not resected because of difficulty in mobilizing it due to adhesions. The removal of the lingula at a second stage was contemplated, but since operation he has at no time had cough productive of purulent sputum.

Case 2. Mrs. B. W., a 41 year old housewife, entered Vanderbilt Hospital August 15, 1941. For three years she had been troubled with cough productive of small amounts of foul sputum, and of small amounts of blood.

Bleeding always occurred at the time of menstruation, and during the last two periods before admission she had profuse pulmonary hemorrhages which left her weak and dizzy.

Physical examination revealed her to be well developed and nourished. The nose and throat and examination of the chest were negative except for slight lag in expansion in the left lower chest. Repeated sputum examinations revealed no tubercle bacilli. Bronchoscopy revealed inflammation of the left lower lobe bronchus and bronchograms showed dilatation and clubbing of the left lower lobe bronchi.

Left lower lobectomy was performed on August 26. Her post-operative course was complicated by an encapsulated empyema which was drained September 13. Her recovery was complete and she is now well.

CASE 3. C. C., a 31 year old white man was first admitted to the Vanderbilt Hospital in May, 1939. For eight years he had had cough productive of foul sputum and frequent hemoptysis. He was found to have a large cavity in the base of the right lung and right lower lobectomy was considered but was refused by the patient. He was re-admitted to the hospital on August 12, 1941. The cough had persisted and had become productive of enormous amounts of foul sputum. For three months he had had increasing dyspnea, and a month before admission he had lost one pint of blood.

The patient had lost about 20 pounds during the three months prior to admission. The output of sputum averaged 500 c.c. daily, purulent and very foul. Lipiodol injection revealed multiple cavities in the right lower lobe.

The patient requested lobectomy. Operation was performed on September 2. His post-operative course was smooth and uneventful until the fifth post-operative day, when he suddenly became dyspneic and cyanotic. Thoracentesis was performed, which yielded 200 c.c. of thin blood-tinged fluid, without relief, and the patient expired while aspiration was being performed. Post-mortem examination was not done.

CASE 4. S. S., a 57 year old white painter, was admitted to the Medical Service Oct. 25, 1941. Since the age of 16 he had had occasional bouts of coughing accompanied by fever and some hemoptysis. For six months prior to admission these bouts had been more frequent and bleeding a little more profuse. The patient coughed up no pus at any time.

Physical examination revealed dullness, suppressed breath and voice sounds and a few scattered rales at the base of the right lung. Sputum examination revealed nothing remarkable. Lipiodol injection showed failure of filling of the lower lobe bronchi. Bronchoscopy revealed a shallow area of ulceration in the wall of the lower lobe bronchus. Biopsy was obtained and this was immediately followed by an alarming hemorrhage. Microscopic sections of the tissue showed no evidence of neoplasm. Six days after bronchoscopy a profuse hemorrhage occurred, the patient losing about 1100 c.c. Frequent blood transfusions were given. During the ensuing week there were repeated hemorrhages, and he coughed up 100 to 400 c.c. of blood at various times. On December 3 he lost 1000 c.c. of blood within an hour. The patient lost ground rapidly and his temperature rose to 102° and 103°. On December 4 he was transferred to the Surgical Service and on December 5 right lower lobectomy was performed. Operation was carried out under considerable stress, as an emergency procedure. Examination of the specimen postoperatively revealed bron-

chiectasis and lobar pneumonia. Careful serial sections failed to reveal a neoplasm. His postoperative course was satisfactory for two weeks, when the patient developed a bronchopleural fistula and an empyema and on December 20, the empyema was drained. A wide-spread infection of the chest wall ensued, which gradually subsided. The bronchopleural fistula persisted, but the empyema cavity became clean and smaller. He gradually lost weight and strength and died Feb. 27, 1942. Autopsy revealed terminal bronchopneumonia, acute pyelonephritis, acute interstitial myocarditis and a bronchocutaneous fistula. The remaining pleural space was small and clean.

CASE 5. Miss M. J. C., aged 29, was admitted to the hospital Feb. 18, 1942. Five years before she had had "pleurisy" on the left side. Since that time there have been recurrent episodes of fever and pain in her left chest.

LOBECTOMIES PERFORMED AT VANDERBILT UNIVERSITY HOSPITAL
AUGUST 1941 - AUGUST 1942
(Suppurative Disease)

Case No.	Age	Color	Sex	Diagnosis	Operation Date	Operation Technic	Postop. Course	Result
1.	12	W.	M.	Bronchiectasis L.L.L. and Lingula.	8-14-41	Dissection of Hilum, L.L.L. Chest Closed.	Small Empyema Drained 3 wks.	Well
2.	41	W.	F.	Bronchiectasis L.L.L.	8-26-41	Dissection of Hilum, L.L.L. Chest Closed.	Empyema Drained 3 wks. Postop.	Well
3.	31	W.	M.	Multiple Abscesses R.L.L.	9-2-41	*Tourniquet* Individual Ligation of Vessels. Chest Closed.	Satisfactory Until 5th Postop. Day	Died Suddenly 5th Postop. Day
4.	57	W.	M.	Bronchiectasis R.L.L. Repeated Hemorrhages.	12-5-41	*Tourniquet* Mass Ligation of Hilum. Chest Closed. Emergency.	Empyema Drained 12-30-41. Persistent Broncho-pleural Fistula.	Died 3 mos. After Operation.
5.	29	W.	F.	Bronchiectasis L.L.L.	2-25-42	Dissection and Individual Ligation. Chest Closed.	Uneventful. No Empyema.	Well
6.	31	W.	M.	Multiple Abscesses L.U.L. Repeated Hemorrhages.	3-10-42	*Tourniquet* Mass Ligation of Hilum. Drained at Operation. Emergency.	Satisfactory (Pleural Cavity Drained at Operation)	Well
7.	29	W.	F.	Bronchiectasis L.L.L.	5-5-42	Dissection and Individual Ligation. Chest Closed.	Satisfactory No Empyema.	Well

Ten months before admission she had entered Nashville General Hospital with a temperature of 104° and was found to have bronchopneumonia, left lower. She subsequently was twice re-admitted to that hospital with bronchopneumonia, eight months and three and one-half months before admission to Vanderbilt, and there was almost constant pain in her left lower chest from the time of the first episode. Bronchograms were made following the last acute illness and a diagnosis of left bronchiectasis was made. Bronchograms were repeated at the Vanderbilt Hospital which confirmed the diagnosis and showed the lesion to be confined to the left lower lobe. Bronchoscopy revealed no evidence of neoplasm. Left lower lobectomy was performed on February 25. Her postoperative course was smooth and without complication, and she left the hospital three weeks after operation. She is now well.

CASE 6. S. M. B., a 31 year old mechanic, entered the hospital on Dec. 31, 1941, on the Medical Service. Two months before, he had gone to bed with high fever, chills, pain under the left scapula and cough productive of blood-tinged sputum. This illness subsided and he returned to work after three weeks. He continued to cough, with the production of increasing amounts of foul yellow sputum. During the week prior to admission cough was productive of bright blood on four occasions, and he had chills on two occasions.

Physical examination showed a stocky obese white male who appeared moderately ill. There was slight impairment of the percussion note over the left apex. No other remarkable findings were present. Repeated sputum examinations revealed fusospirochetal organisms and cocci in chains. No acid-fast organisms were found. X-ray examination showed a cavity in the left upper lobe suggestive of lung abscess. He was sent home on postural drainage. He failed to improve, continued to have irregular bouts of fever and copious amounts of foul sputum. He was re-admitted to the hospital Feb. 3, 1942. Conservative therapy (postural drainage, neoarsphenamine and sulfonamide therapy) again failed to bring about improvement. He was transferred to the Surgical Service and on February 23, one stage drainage of the lung abscess (three pockets were opened) was accomplished. His convalescence was uneventful for ten days, when he again began to have fever and to bring up increasing amounts of foul sputum. On March 8 he suddenly coughed up about an ounce of bright blood. This was repeated several times the following day and on March 10 he coughed up about 200 c.c. of blood. This was followed, within an hour, by two profuse hemorrhages. There was no bleeding from the wound. It was felt that an attempt should be made to provide external drainage or to excise the bleeding lung. He was taken to the operating room and exploration of the chest performed. Rather profuse bleeding, into the bronchus, occurred during operation. The entire upper lobe was mobilized as quickly as possible and total lobectomy, using the tourniquet technic, was performed. His postoperative course was satisfactory. He recovered and is now well.

CASE 7. Mrs. E. V., 29 years old, was admitted to Vanderbilt University Hospital on May 5, 1942. For six years she had had occasional hemoptysis. Two years previously she had been found to have chronic maxillary sinusitis and she was admitted to the hospital for bilateral Caldwell-Luc operation, which was performed. The respiratory symptoms continued without improvement and bronchograms showed dilatation and clubbing of the bronchi, limited to the left lower lobe. She had lost 45 pounds during the six years of her illness.

Physical examination revealed no abnormal signs except evidence of malnutrition and inconstant sticky rales at the left base.

Left lower lobectomy was performed on May 16. On the seventh postoperative day she became dyspneic and there were evidences of a large fluid collection in the left chest. This was drained, about 1500 c.c. of thin, sterile, blood-tinged fluid being removed. Her course was otherwise uneventful and she left the hospital three weeks after lobectomy was performed. She is now well.

This small group of patients is not presented as being of statistical value. The following points are of interest, however:

1. Two patients were operated upon as "emergencies." It was believed that neither patient would survive without these procedures. One of these patients lived for three months after operation. The other is alive and well at this time.

2. All patients with uncomplicated bronchiectasis (four patients) on whom planned procedures were carried out are symptom-free at this time.

3. There was only one persistent bronchial fistula (the patient who died three months after operation.) All others are completely healed.

4. The chest was closed without drainage in all but one case (Case 6). It is believed that primary drainage should have been provided in cases 3 and 4, and that closure of the chest, without drainage, is proper following lobectomy for uncomplicated bronchiectasis.

REFERENCES

1. Bradshaw, H. H.: Putney, F. J., and Clerf, L. H.: The Fate of Patients with Untreated Bronchiectasis, J. A. M. A. *116:* 2561, 1941.
2. Perry, K. M. A., and King, D. S.: Bronchiectasis: A Study of Prognosis Based on a Follow-up of Four Hundred Patients, Am. Rev. Tuberc. *41:* 531, 1940.
3. Riggins, H. M.: Bronchiectasis: Mortality and Morbidity of Medically Treated Patients. Am. J. Surg., *54:* 50, 1941.
4. Ogilvie, A. G.: The Natural History of Bronchiectasis, Arch. Int. Med. *68:* 395 1941.
5. Churchill, E. D.: Bronchiectasis: Physical and Psychological Manifestations. New Eng. J. Med. *218:* 97, 1938.
6. Neuhof, H., and Wessler, H.: Putrid Lung Abscess, Its Etiology, Pathology, Clinical Manifestations, Diagnosis and Treatment, J. Thor. Surg. *1:* 637, 1932.
7. Shaw, R. R.: Pulmonary Abscess, J. Thor. Surg. *11:* 453, 1942.
8. Archibald, E.: Le Traitement Chirurgical des Suppurations Pulmonaires, Arch. Med. Chir. de L'appar. Respir. *2:* 411, 1927.
9. Alexander, J.: Total Pulmonary Lobectomy, Surg., Gynec. & Obst. *56:* 658, 1933.
10. Churchill, E. D.: Resection of the Lung. Surgery. *8:* 961, 1940.

LATRODECTUS MACTANS (BLACK WIDOW SPIDER) BITE AS A SURGICAL PROBLEM

K. H. AYNESWORTH, M. D.
Waco, Texas

ALMOST every year, especially during the early summer and fall, patients have been brought into the Providence Hospital suffering from severe, acute abdominal and general symptoms which were difficult to diagnose because of the confusion of the general symptoms with what appeared to be definite symptoms of some acute surgical condition within the abdomen. The abdominal symptoms, while often obscure, seemed to dominate the diagnostic picture. Several of these patients were subjected to needless operations, generally with the provisional diagnosis of acute appendicitis or, in one instance, a ruptured viscus. In no case was anything found to justify either the diagnosis or the surgical intervention. The error was not limited to the younger and less experienced surgeons; indeed the majority of operations were performed by the more experienced surgeons. Candor in most instances compelled the operator to say that the findings failed to justify the operative procedures. Generally, these patients were brought to the hospital by the family physician who urged operation.

One of the reasons that this subject is discussed at this time is because, with so many soldiers living in camps, the incidence of black widow spider bite is apt to increase. It is my purpose to remind surgeons that the signs and symptoms following black widow spider bite closely simulate those of acute surgical conditions of the abdomen and to stress the importance of carefully taken histories. If this can be impressed upon surgeons many needless operations may be prevented.

The literature of Latrodectus mactans poisoning is voluminous and from almost every country. I have read articles from every country in Europe, Asiatic Russia, South Africa, Australia and, in fact, from many other lands. It seems that the sting of this spider has been known for centuries and has produced a literature in the scientific journals as well as in the folklore of the peoples everywhere. Recently, scientists have been making a study of the Latrodectus mactans, nowhere more accurately than in the Southern section of the United States, as many of the best articles have come from this section. I have not tried to abstract the literature, but have cited only a few of the more recent articles, to illustrate the clinical phase of the subject. The bibliography herewith presented

Presented before the Texas Surgical Society, Oct. 5-6, 1942, at San Antonio.

is only for those who wish to pursue the subject further. It is an extremely interesting subject.

Among the earlier reports on the poison of the Latrodectus mactans was a paper published by Bogen[1] in 1926, in which he went into great detail about the toxic effects of the spider bite and gave many references to other reports. His was the first classic description of the symptoms, with the many variations and unique manifestations and the multiform symptomatology of the intoxications. Since then, the medical profession has been put on notice as to the effects of Latrodectus mactans poisoning. Unfortunately, many surgeons either did not read this paper or did not remember its lessons when such patients were brought to them. The literature is replete with reports of the mistakes since then. When the major symptoms occur, which simulate so closely an acute surgical condition of the abdomen, the patients are hurried to the hospital for operation. Being uninformed of the suspected spider bite, the surgeons emphasize the acute abdominal symptoms, overlook the general symptoms, which are always present, and advise operation. A recent case in my practice will illustrate the complexities of both diagnosis and treatment.

Report of Cases

Case 1. The patient was 72 years of age, a chronic sufferer from indigestion of a painful, recurring type, associated with spells of intermittent diarrhea. He was overweight, very feeble and practically blind. The history was as follows: He went to the toilet outdoors for a bowel movement. While there, he noticed a stinging sensation on the scrotum, but paid little attention to the slight pain. He returned to the house and almost immediately began to feel very sick and to have moderate but generalized pains all over the body. The bowels acted several times. He began to feel nauseated and shortly thereafter vomited a few times. There was notable restlessness and much general irritability. He spoke incoherently from time to time. He began to complain of very severe pain in the abdomen, which at times came in colicky attacks, during which he would roll and toss about the bed. The location of the greatest pain was in the right side of the epigastrium and down the right side of the abdomen. A physician was called who, after examining him, made a diagnosis of acute appendicitis. To relieve the pain paregoric was given him, but with poor results. Then a rather large dose of morphine was administered, which quickly relieved the major pain and he went to sleep.

This case history illustrates the typical symptomatology of gastric or duodenal perforation. The physician who referred the patient to the hospital sent a note stating that he was suffering from acute appendicitis and advising immediate operation. The patient was then under the influence of morphine and could not answer questions intelligently. The problem before the surgeon was to secure a correct history of the beginning of the illness. Above all, it was necessary not to overlook the possibility of a ruptured gastric or duodenal ulcer, or even acute appendicitis.

At this time the abdominal symptoms almost completely overshadowed the general symptoms and the patient was unable to give a history of the onset of the disease. Examination of the abdomen clearly suggested an acute surgical condition, probably a ruptured viscus. Still, there was another possibility, a ruptured gallbladder, especially since the patient had suffered from epigastric symptoms. The entire picture was that of a serious upper abdominal condition. The main symptoms were acute, severe, constant pain in the right epigastrium, nausea and occasional vomiting. The right rectus was board-like, tender and fixed. There was a greatly distended abdomen, especially in the upper half. There was extension of muscle rigidity and tenderness down into the right iliac fossa. There was marked tympany, but the liver dullness had not disappeared. The heart and lungs were normal. The diaphragmatic actions were limited, especially on the right side. The massive doses of morphine made the patient stupid and quiet. When first aroused he was somewhat restless, but would move with great caution, often holding his hand on the abdomen. When completely aroused, he would become more active and move about with freedom.

The general symptoms of pain, muscle stiffness and tenderness were still present but much less noticeable. The laboratory findings were those of increased total blood count with high differential. The urine was normal.

The question now was what should be done? A diagnosis of some general toxic poisoning was made and the patient given sedatives. He left the hospital four days later in good condition. The diagnosis was made on the basis of symptoms—and nothing else.

CASE 2. This is the case of a young farmer, who gave the following history: Shortly after eating a hearty dinner, he was seized with general cramping of the muscles of the arms, legs, and abdomen, and with a general spastic condition of the musculature of the entire body. In addition, he suffered from severe general body pains, which were at times apparently paroxysmal. He was able to walk, although with some difficulty.

A physician was sent for who, after examination, made a diagnosis of acute appendicitis, and advised operation.

On arrival at the hospital, the history disclosed that the patient had been in good health previously. Before taking his customary nap, he went to the closet, an outdoor affair, and had a bowel action. Almost immediately he was taken with violent cramps all over, most severely in the abdomen, with milder cramps in the muscles of the arms, legs and generalized body pains. He vomited twice early in the attack without relief of symptoms. He was restless and moved about with jerky actions.

On admission he had a fever of 100° and a pulse rate of 110. The blood findings showed a high leukocyte count and a high differential, but there was nothing in the blood picture peculiar to Latrodectus mactans poisoning.

Physical examination disclosed a rigid, fixed abdomen, especially in the lower right quadrant. There was definite local tenderness, but no cutaneous hyperesthesia. Tympany was normal. No mass could be felt. Otherwise, with the exception of a general muscle tension and moderate tenderness, the abdomen was normal.

As has been my custom, I told the internes and the resident to examine the patient and make a diagnosis. Acute appendicitis was diagnosed.

After considerable discussion, I suggested that further attention be given to the preliminary symptoms which the patient could supply. While the ab-

dominal symptoms strongly suggested an acute inflammatory disease, as did the local examination, there were other factors to be considered before a decision to operate should be made. Among these were generalized, though moderate, muscle tension in the arms, legs and chest, and moderate muscle tenderness. The muscle action was perfect but limited by tenderness. The joint actions were normal. The reflexes were exaggerated. The evidence showed generalized spinal irritation both in the motor functions and in the sensory system. Since the abdominal muscles are the best conditioned for the display of spasm, being long and flat and always in tension, the greatest evidence of spinal irritation would be shown in the abdominal muscles. At times the spasms of pain would be distinctly paroxysmal. In addition, there was a reaction to some toxin as shown by nausea, vomiting, muscle pains, increased reflexes and mental confusion and, further, by fever, increased blood reactions, etc.

During the conference, every detail of diagnosis of the acute surgical abdomen was investigated. Finally, when the decision was made not to operate, the patient roused up, remembered having been bitten on the scrotum by a black spider. Then the situation was clear. No one present at that time had ever heard of the Latrodectus mactans. The decision not to operate had been made after a study and analysis of the inaugural symptoms, the development of the symptoms and the physical examination.

The two case reports given above are sufficient to illustrate the necessity for a scientific study of the symptoms before operating for supposed acute abdominal diseases. A number of similar cases can be reported from the same hospital. Unfortunately, three of these patients underwent appendectomies. My two cases are typical of many others which make the diagnosis of some acute surgical emergency a problem of logical deduction, inference and information. A careful study of the histories of these two patients shows that they were suffering from some toxin, or other poisoning, which affected the entire neuromuscular system. It is difficult to understand why there were spasms of the skeletal muscles without at the same time similar spasmodic reactions of the intestinal muscles if the spasms were caused by a toxin distributed throughout the body fluids and the vascular system to every muscle in the body. Probably, the autonomic and the sympathetic nervous systems are not activated as are the skeletal muscles. In both of these patients, there were localized areas of abdominal muscles which were in extreme spasm, even rigidity, sufficient to cause imitation of true muscle rigidity produced by inflammatory reaction—one in the region of the duodenum, the other in the region of the appendix. Yet, a carefully taken history of the illness would not permit the diagnosis of an acute surgical condition of the abdomen.

According to the reports of the pathologist in Providence Hospital, normal appendices are removed more often than should be in a modern hospital where all the facilities for diagnosis are at hand. And I am convinced that this is true in most hospitals. Yet, statistics show that acute appendicitis alone causes the death of

more than seventeen thousand people annually, most of whom would not have died had the diagnosis been made before complications developed. The same criticism may be made concerning the other acute surgical diseases of the abdomen. Which brings me back to the importance of histories.

The study of the symptoms of Latrodectus mactans poisoning is very confusing to the average physician. It is very easy to acquire a laissez-faire attitude in examining a patient, especially a patient who presents symptoms of acute abdominal pain. The dominating symptoms in patients with acute surgical conditions of the abdomen are pain, nausea and vomiting associated with a rise in temperature, and an increase in pulse rate. When such a complex is presented to the physician, acute appendicitis is the first idea which comes to his mind. Inasmuch as these are, also, the first symptoms which the arachnidism patient gives the examiner, failure to consider associated symptoms and to evaluate them in the order of their importance is quite easy. It is the usual rule.

There is another pitfall: it is not unusual for patients suffering from real acute, surgical conditions within the abdomen to have preliminary general symptoms which are the characteristics of Latrodectus mactans poisoning. But, an analysis of the preliminary symptoms should show that they are the most important ones in the diagnostic complex in arachnidism. The patient who has suffered Latrodectus mactans bite presents a diagnostic picture of a general toxemia before he develops the local manifestations of disease in the abdomen. Also, the abdominal symptoms are an expression of a general condition rather than the reverse. Then there are other phenomena in the diagnostic picture which should have serious attention: the patient displays the symptoms of acute spinal irritation in every muscle of the body, including the respiratory musculature. There are in many patients such evidences of an acute disturbance of the sensorium as delirium, stupor, etc., which are not the usual accompaniment of acute abdominal diseases. The presence of these general symptoms should arouse the suspicions of the examiner.

In all of my patients, the evidences of general spinal irritation kept me from a hasty diagnosis of the abdominal symptoms as a surgical emergency. It is true that there were the typical symptoms of acute abdominal disease. Among these symptoms were acute colicky or paroxysmal pains, diffusion of the pain to various regions of the abdomen, localized muscle rigidity in some instances, which ordinarily would indicate acute appendicitis, a ruptured duodenal or gastric ulcer or some other surgical emergency. In addition, there was nausea or vomiting or both in every one of them. With this

local picture, one would seriously consider the most likely disease rather than think of some general toxemia to explain the presenting symptom complex. The final correct summing-up of all of the symptoms depends upon the history of the ailment from the onset, rather than from the major presenting symptoms when the surgeon sees the patient. In no instance have I operated upon one of these patients. Unfortunately, three of them were operated upon in the hospital in which I do the major part of my work. This, too, in the face of a full discussion of the subject before a staff meeting previously.

In the present war time, when many soldiers are subjected to the possibility of Latrodectus mactans poisoning, the subject is of sufficient significance to present to this Society.

BIBLIOGRAPHY

1. Bogen, E.: Arachnidism. Study in Spider Poisoning, J. A. M. A. *86:* 1894, 1926.
2. Walsh, G.: Arachnidism: Case of Poisoning from Bite of Latrodectus Mactans, South. M. J. *23:* 1038, 1930.
3. Walsh, G., and Morgan, W. G.: Arachnidism: Report of a Series of 29 Cases of Poisoning from the Bite of the Latrodectus Mactans, Am. J. M. Sc. *186:* 413, 1933.
4. Gilbert, E. W., and Stewart, C. M.: Effective Treatment of Arachnidism by Calcium Salts, Am. J. M. Sc. *189:* 532, 1935.
5. Frawley, J. M., and Ginsburg, H. M.: Diagnosis and Treatment of Black Widow Spider Bite, J. A. M. A. *104:* 1790, 1935.
6. Hargis, A. S.: Arachnidism, New England J. Med. *215:* 489, 1936.

SAFETY VALVE ANASTOMOSIS AND DECOMPRESSION IN INTESTINAL SURGERY BY USE OF A "T" TUBE

GEORGE L. CARRINGTON, A. M., M. D.
Burlington, N. C.

THE purpose of this paper is to report the use of a "T" tube in four cases of intestinal surgery, to discuss some of the problems involved in the type of cases in which it has been employed by me, and to suggest that it may have a wider applicability. In two cases it was used as a safety valve at the site of anastomosis, and in two cases it was used for decompression only.

The procedure was first employed by me in October, 1941, upon a young man with an intestinal obstruction of six days' duration. At that time the small intestines were tremendously distended and showed no peristalsis. It was necessary to resect four and one half feet of the terminal ileum. An end to end anastomosis was performed one inch from the ileo-cecal valve. It was done about a "T" tube, the short arm in the lumen of the gut being about one and a half inches long. Fine chromic catgut was used for the anastomosis—two layers continuous chromic 5-*o* and one layer chromic *o*. The gut was brought close to the peritoneum but not sutured to it. The long arm of the tube was connected to a Wappler-Stedman suprapubic pump and light intermittent suction applied. The wound was closed in layers about the long arm of the "T".

His postoperative course was uneventful. An indwelling nasal tube was used for 48 hours to keep the upper gut decompressed. He received 5 per cent glucose in Ringer's solution by continuous venoclysis at the rate of 3000 c.c. every 24 hours to keep up his sugar and electrolytes, and a blood transfusion to maintain blood serum and oxygen-carrying capacity of the red cells. The "T" tube came out easily when tested twelve days after operation. There was no fistula. After two days there was no staining of the dressing. He has been well since.

The second patient was a middle-aged colored woman with late obstruction due to volvulus and a chewing gum band. I released the obstruction, but felt dubious about the ability of the distended gut to regain tonicity. The obstruction again was in the terminal ileum. I decided to insert a "T" tube in the gut and maintain intermittent light suction on it. With an indwelling nasal tube and maintenance of fluids she made an uneventful recovery. The tube was removed after 12 days and the wound again closed with very little drainage.

The third patient was a middle-aged white man with a constricting tubercular lesion in the first portion of the transverse colon. I resected the cecum, ascending and first portions of the transverse colon, then did an end to side anastomosis of ileum to colon, and did it about a "T" tube, the short arm of which was about an inch and a half long. Several days elapsed before drainage was obtained. Better results would probably have been secured with a shorter segment of tubing in the gut, as the anastomosis here was a right angle affair. The tube was removed after twelve days, and again there was no fecal drainage, merely a stain for two days.

The fourth patient was a six year old boy who after appendectomy developed an obstruction due to edema in the gut wall. The cecum and about two inches of the terminal ileum were edematous and without peristalsis. The gut was distended above that area. Just above the area of edema I made a small stab wound in the ante-mesenteric border of the gut and inserted a "T" tube with the short arm cut down to one and one fourth inches. The gut was brought against the peritoneum but not sutured. The wound was closed about the long arm of the tube. In twelve hours the tube was draining well into the bottle connected with the suction system and the patient appeared out of danger. The tube was removed eleven days after operation and he was discharged on the twelfth day without fecal staining.

The problems of intestinal resection with anastomosis and those of late obstruction with or without resection and anastomosis are closely related. Most resections present an obstruction for several days after operation. The obstruction may be either functional or mechanical. Bruising of the gut, inflammation at site of operation and interference with innervation, may result in a functional ileus. Edema at and around the suture line may produce a mechanical obstruction. Several years ago I found the stoma of a small gut anastomosis stuck together with fibrin when it was necessary to reopen the belly a few days after the original operation because of persistence of obstructive symptoms.

This finding, the fact that most of my patients with small gut anastomoses take about four days to recover from obstructive symptoms, and the memory of the excellent results reported by some of the older surgeons with the use of Murphy buttons suggested the "T" tube at the site of anastomosis. It seemed a better procedure than ileostomy above the anastomosis, which I had at times done, since it would both decompress the gut, relieve tension on the suture line, and maintain the patency of the stoma.

In the instances that I have used this procedure it has been possible to have the tube near the lateral wall of the abdomen. This

has added to the safety factor and reduced the chances of secondary obstruction around the tube. In one instance I was able to employ omentum as an additional safeguard.

In the third case, where resection of the colon was anticipated, the only one of the four not operated upon because of obstruction, the patient received sulfaguanadine for several days before operation.

So far I have not had occasion to use the procedure as a substitute for a so-called Mikulicz type of resection, but believe that at times it might be preferable.

Devine's[1] excellent operation of defunctioning the distal colon greatly increased the safety of resections of portions of that gut. The length of hospitalization, however, for that procedure is rather great—though so far in my opinion well justified. Recently, I have been wondering if the resection of lesions in the distal colon could not be done safely without defunctioning and with immediate anastomosis around a "T" tube to relieve tension on the suture line.

Summary

Four cases of the use of a "T" tube in intestinal surgery are reported. In two instances the tube was used simply to decompress the obstructed gut. In the two other cases it was used at the site of anastomsis for the triple purposes of decompressing gut, relieving tension on the suture line and maintaining the patency of the stoma. The suggestion is made that it may also find usefulness in surgery involving the left half of the colon. "Safety valve" or "decompressive" anastomosis would be appropriate descriptive terms for the procedure when used in anastomoses.

REFERENCE

1. Devine, Sir Hugh: Operation on a Defunctioned Distal Colon, Surg. *3:* 165-194 (Feb.) 1938.

THE LATERAL MIGRATION OF THE APPENDECTOMY INCISION

Its Relationship to Mortality

RICHARD E. STRAIN, M.D.
Nashville

CATHARTICS, pathology in the appendix, drainage, age and time are fairly well recognized factors in the mortality of appendicitis. The location of the incision and its relationship to the mortality is not yet appreciated by a great many surgeons. Use of sulfonamides intra-abdominally gives promise that the penalty for use of a poorly chosen avenue of approach to the area of infection in appendicitis may not be so great in the future as in the past, but their use should not serve as an excuse for poor technic.

The earliest account of an operation for appendicitis by Hancock[1] reported the use of a far laterally placed incision. The patient was operated on ten days following delivery and seven days after onset of pain. "The patient was put under chloroform and an incision about four inches long made inwards from the spine of the ilium above Poupart's ligament, but as close to it as possible. Upon opening the abdomen, a quantity of excessively offensive turbid serum, with fibrinous floculli, poured out mixed with air globules and also patches of false membrane." Fifteen days postoperatively a "ball of fecal matter surrounded by calcareous deposit" was removed from the wound and recovery was then rapid.

In 1867 Willard Parker[2] urged operation and reported success in three out of four cases. Early operation was advised at the time but the type of incision used is not stated. Bull,[3] while not recognizing the true condition of appendicitis, urged operation for perityphlitis and used "an incision six inches long parallel to Poupart's ligament, over the middle of the iliac fossae." Homans[4] opened a "perityphlitic" abscess "over the most tender point which was one and a half inches above and a little posterior to the right anterior superior spine of the ileum."

In October, 1886, Fitz[2] presented his masterly report on appendicitis but did not recommend any type of incision for operation. In 1889 McBurney[5] reported on McBurney's point and stated he and Dr. Sands were using "a slightly oblique incision four and a half inches long, the center of this incision being two inches from the anterior superior spine toward the umbilichus. The incision should be a liberal one, for much room may be required, and a five inch cut in the adult is not too much. It should follow as nearly as possible the right edge of the rectus muscle."

In 1894 McBurney[6] reported on his muscle splitting incision which Strohl[7] states was used by McArthur for three years on sixty cases before McBurney used it. McBurney described the incision as crossing "a line drawn from the anterior iliac spine to the umbilichus nearly at right angles about one inch from the iliac spine." External oblique, internal oblique and transversalis fascial fibers were separated or split and not cut. The incision in the peritoneum was transverse. McBurney also stated the incision could be enlarged by continuing separation of the fibers to the rectus sheath and ileum. Harrington[8] first proposed "enlarging the McBurney incision for interval cases to obtain sufficient opening to treat a large proportion of the acute inflammatory cases" by separating the fibers of external oblique, internal oblique and transversalis muscle "from ileum to the linea semilunaris. If this does not give sufficient room, the sheath of the rectus may be separated in front and behind the muscle in a line continuous with the separation of the internal oblique and transversalis muscle." Weir[9] popularized this extension of the McBurney incision. McBurney did not appreciate the relationship between mortality and a laterally placed incision for he stated "This operation does not appear to be suitable for cases accompanied by suppuration about the appendix, which require to be treated by extensive packing with gauze."

Battle[10] in 1895 operated on an eighteen year old boy through an incision which was "one and a half inch to the inside of the linea semilunaris and divided the aponeurosis of the external oblique with the sheath of the rectus. The rectus muscle was then drawn to the inner side, and the posterior layer of the sheath and transversalis fascia exposed, the inner incision not corresponding to the external. The peritoneum was then divided." Mr. Battle felt this incision "absolutely prevented hernia." Lennander[11] modified Battle's incision by going directly through the rectus muscle. Edebohls[12] did much to popularize this incision.

The transverse incision was first reported by Elliot.[13] He stated, "I begin the operation by making a horizontal cut through the skin and the aponeurosis of the external oblique, beginning one half inch inside the anterior superior spine of the ileum and extending to the linea semilunaris. The fibres of the external oblique are thus cut across but the fibres of internal oblique and the transversalis are separated as in McBurney's operation. This incision may be enlarged upward or downward in the linea semilunaris or may be extended into the rectus if necessary." Davis[14] emphasized the use of Elliot's incision.

Some men have used a midline incision, McClure and Altemeir[15] report a mortality of 27 per cent in eleven cases in which a midline incision was used and urge the value of a McBurney incision.

Thus we see the incision for appendectomy has migrated from Hancock's original one placed far laterally to the midline and again back to a position slightly medial or slightly lateral to the linea semilunaris. This medial migration of the incision which is fraught with such danger of opening the general peritoneal cavity and spreading the area of infection was opposed by Sir James Berry[16] when he stated, "It is a matter of the greatest importance that the incision be made through the area of adhesions only. . . . Incisions in the semilunar line or through McBurney's point will usually also open the non-adherent peritoneum."

In recent years many writers[17] have emphasized in their series that the McBurney incision carries a lower mortality than other types of incisions more medially placed yet some recent texts[18] say the incision should be abandoned. Rumbold[19] found the right rectus or paramedian incision carried a mortality rate almost four times that of the McBurney incision in the less complicated cases and over twelve times the latter rate in graver forms of the disease. These men emphasize that extensive manipulation is not done, so infection is not spread. I have felt more emphasis should be laid on the fact that tension is relieved and decompression accomplished in the area of infection also. When one opens the peritoneum and is greeted with a gush of turbid fluid it is obvious that not only has a "vent" been provided which aids in preventing rupture through the protecting wall of adhered bowel and omentum but tension, which is one of the important factors concerned in absorption of toxins as well as in spread of infection, has been released. It would do little good to relieve this tension and release this infectious material if it were allowed to spread throughout the peritoneal cavity. Under these conditions operation should be done as soon as the diagnosis is made *only if a far laterally placed* incision *is used.* It is my belief that those men who use a right rectus incision or one close to the semilunaris certainly should not operate on so-called "late" cases as they will surely disseminate the infection. Babcock[20] has accomplished "decompression" in patients too sick for operation by making a stab wound within the area of infection and applying continuous suction through perforated glass or alloy steel tubes. Hancock[1] felt the "typhoid condition into which patients affected with peritoneal inflammation fell did not depend upon the violence of the disease but upon the acrid nature of the effused fluid, the removal of which he thought the only chance of saving the patient."

Probably one important aspect of the incision for appendicitis needs emphasis. From the view of dissemination and mortality the important point is not so much where the incision is made through muscle or fascia but *where the peritoneum is incised in relation to the area of infection.* The peritoneal cavity may be opened on its lateral or even posterior aspect through a right rectus incision if one desires.

For five years I have adopted the following routine*—immediate operation as soon as the diagnosis is made regardless of the time element; transfusion in all serious cases; a short wait (two to three hours) to replenish fluids in badly dehydrated cases. I have used an oblique muscle splitting incision placed one finger breadth medial to the anterior superior spine. As the peritoneum is encountered one slides down its lateral wall extraperitoneally a short distance; the peritoneal cavity is entered on its lateral wall in an oblique direction. The appendix is approached by gently pulling the cecum laterally with non-traumatizing forceps or, in a retrocecal case, by rolling the cecum medially. In cases in which frank extraperitoneal edema and a glazed peritoneum covered with fibrin shows the area ventral to the appendix is already involved, I incise the peritoneum on its anteriolateral aspect and slide down the lateral wall within the peritoneal cavity.

This approach seems to have the advantage that the incision through peritoneum stays out of the peritoneal cavity medial as well as ventral to the appendix. It also offers the shortest possible route of access to the appendix without danger of extensive spread of infection. I have operated on forty-nine consecutive cases of acute perforated appendix, classifying cases as acute, acute gangrenous, acute perforated and acute with abscess, by this method with one death, this patient having bilateral lobar pneumonia with a ruptured appendix on admission. Death occurred twelve hours after admission. A majority were operated on before sulfonamides were in general use and none of them received these drugs. The appendix was removed in each instance. Drainage was used in each. The drain was moved daily to prevent its acting as a plug and drainage was continued as long as discharge was present. No hernias were noted but in two cases the granulating adherent skin, external oblique, internal oblique and transversalis were dissected free and secondarily closed to prevent hernias by a method similar to Edebohl's[12] except that Edebohl cut peritoneum while I made a point of not entering the peritoneal cavity if possible to avoid such entry.

---

*Work done at the Huchow, China, General Hospital and The Miraj Medical Centre, Miraj, India.

SUMMARY

The lateral migration of the appendectomy incision from the midline to the region of the anterior superior spine has been accompanied by a steady decrease in mortality.

The important point about any appendectomy incision as regards dissemination and decompression, whether muscle splitting or otherwise, is not where the incision divides muscle or fascia but where, in relation to the area of infection, the incision enters the peritoneal cavity.

Forty-nine consecutive cases of acute perforated appendices have been decompressed and appendectomy performed through a muscle splitting incision which enters the peritoneal cavity lateral to the area of infection.

All cases were drained.

Sulfonamides were not used.

One death was noted.

BIBLIOGRAPHY

1. Hancock, Mr.: Disease of Appendix Caeci Cured by Operation, Lancet *2*:380-382, 1848.
2. Fitz, R.: Perforating Inflammation of the Vermiform Appendix, Am. J. M. Sc. *92*:321-346, 1886.
3. Bull, W. T.: Perityphlitis (Article II), New York M. J. *18*:240-264, 1873.
4. Homans, J.: A Successful Case of Laparatomy for Perityphlitic Abscess, Boston Med. and Surg. J. *114*:388-389, 1886.
5. McBurney, C.: Disease of the Vermiform Appendix, New York M. J. *50*:676-684 1889.
6. McBurney, C.: The Incision Made in the Abdominal Wall in Cases of Appendicitis with a Description of a New Method of Operating, Ann. Surg. *20*:38-43, 1894.
7. Strohl, E. L.: Appendectomy by the Muscle Splitting Technic, Surg. Clin. North America *22*:1-8 (Feb.) 1942.
8. Harrington, F. B.: Hernia Following Operation for Appendicitis, Boston Med. and Surg. J. *141*:105-108, 1899.
9. Weir, R. F.: An Improved Operation for Acute Appendicitis or for Quiescent Cases with Complications, Med. News *76*:241-242, 1900.
10. Battle, Mr.: Modified Incision for Removal of the Vermiform Appendix, Brit. M. J. *21*:1360, 1895.
11. Lennander, K. G.: Ueber den Bauchschnitt durch eine Rectusscheide mit Vershiebung des Medialen oder Lateralen Ravdes des Musculus Rectus, Zentralbl f. Chir. *25*:90-94, 1898.
12. Edebohls, G. M.: The Hernia Guarantee and the Minimum Confinement After Operation for Appendicitis with and without Pus, M. Rec. *55*:665-667, 1899.
13. Elliot, J. W.: A Modification of the McBurney Incision for Appendectomy, Boston M. & S. J. *2*:433-434, 1896.
14. Davis, C. G.: A Transverse Incision for the Removal of the Appendix, Ann. Surg. *43*:106-110, 1906.
15. McClure, R. D., and Altemeir, W. A.: Acute Perforated Appendicitis with Peritonitis, Ann. Surg. *105*:800-813 (May) 1937.

16. Berry, Sir James: Early vs. Late Operations in Cases of Inflamed Appendix, Lancet *2:*1857-1858, 1899; Operations for Acute Appendicitis, Lancet *1:*197, 1900; "Fallen Idols," The Case of Appendicitis, Lancet *1:* 1027-1030, 1932.

17. Lehman, H.: Die Appendicitis Acuta in Greisenalter, Wien. Klin. Wchnschr. *40:*995-997, 1927; Muller, G. P.: The Acute Appendicitis Problem from the Surgical Standpoint, J. Med. Soc. N. J. *37:*587-591 (Dec.) 1940.

18. Thorek, M.: Operative Surgery, Vol. 3, p. 1467, J. B. Lippincott Company, 1938.

19. Rumbold, L.: Some Factors in a Lowered Mortality Rate for Acute Appendicitis, Arch. Surg. *45:*25-54 (Jan.) 1941.

20. Babcock, W. W.: A Better Technic in Operating for Appendicitis, J. Internat. Coll. Surgeons *4:*155-159 (April) 1941.

The Southern Surgeon

Published Monthly by
The SOUTHERN SURGEON PUBLISHING COMPANY
701 Hurt Building
ATLANTA

*L. MINOR BLACKFORD, M.D.
Editor

B. T. BEASLEY, M.D.
Managing Editor

WALTER G. STUCK, M.D.
Associate Editor

*J. DUFFY HANCOCK, M.D.
Associate Editor

H. EARLE CONWELL, M.D.
Associate Editor

*In Active Military Service

Subscription in the United States, $5.00

VOLUME XI NOVEMBER, 1942 NUMBER 11

PHYSICIAN SHORTAGE

It is appropriate and timely that the exact status of the medical profession during war be published, particularly with reference to the number of physicians in this country at present as compared with the population as a whole.

At present there are approximately 176,000 physicians serving a population of approximately 130,000,000 people or one physician to 738 people. (It has been estimated that a ratio of one physician to 1500 people is not too low to give fairly adequate medical service).

An army of 10,000,000 men will require 65,000 doctors or a ratio of 6.5 doctors for each thousand troops. Add to the sixty-five thousand doctors taken into the army 21,000 who will do no practice for one cause or another and there will be 86,000 not available for civilian practice. This number taken from the total medical population will leave 90,000 available for practice or one doctor to 1,333 population. There are approximately 3,500 deaths annually in the medical ranks, and approximately 5,000 new doctors graduated, or 1500 greater number than the death rate.* If only 50 per cent of this number enters practice there will be 750 to take care of the 1,000,000 increase in population or 1 doctor to 1,333 population increase.

These data give a fairly true picture of the status of our profession, and should dismiss from our minds the fear of a physician

*According to present plans the medical colleges will graduate an additional 5,000 in three years as a result of the accelerated-step-up in their curricula, thus adding to the number of doctors 20,000 instead of 15,000 during the next three years.

shortage in this country. This country has more physicians to population by 50 per cent than any other nation. On the basis of world distribution of physicians this country could spare half of its doctors without a shortage.

There is a shortage in certain localities, due to economic and industrial dislocation of population. In many rural communities the doctors moved out, usually to the cities, before the war, and since the war began others have volunteered into the army. Industrial plants have grown up over night in certain localities attracting thousands of employees where there are no doctors. These shortages are relative and can be remedied by proper distribution of the medical profession. Many cities are over-crowded with doctors, and can afford to spare 25 to 50 per cent of their number to the armed forces and to the localities where there is a shortage.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

TEXTBOOK OF CLINICAL PARASITOLOGY, Including Laboratory Indentification and Technic. By DAVID L. BELDING, M. D., Professor of Bacteriology and Experimental Pathology, Boston University School of Medicine; Member of Staff of Evans Memorial and Massachusetts Memorial Hospitals. 888 pages, with 1,438 illustrations. Price $8.50. New York and London: D. Appleton Century Company, 1942.

At no other time in the history of American medicine has the knowledge of clinical parasitology been so essential. Distribution of the armed forces to tropical and subtropical countries, and the evacuation of civilian populations who have lived for years in infested areas in the Eastern hemisphere have brought new problems in the diagnosis and control of parasitic infections which were formerly rarely encountered. The publication of a textbook which presents recent advances in the treatment of parasitic infections and which utilizes unusual and concise technics in presenting essential information is especially timely and helpful. The author is professor of bacteriology and experimental pathology in Boston University School of Medicine and a member of the staff of Evans Memorial Hospital and Massachusetts Memorial Hospital. With this combination of clinical experience and academic background, he is well equipped to discuss parasitic infections in a scholarly way from the viewpoint which appeals to the practicing physician.

This volume combines most of the essential medical information in the three fields of parasitology, namely protozoology, helminthology, and entomology. Pathology, diagnosis, treatment and prevention of the disease take precedence over zoology of the parasite and the textual discussion of these aspects is supplemented by tables and maps. Involved life cycles in most instances are clarified by diagrams. The text is profusely illustrated with drawings and photographs.

The book is divided into seven sections; the first section treats of general parasitology and includes two very interesting chapters on the Immunity of Parasitic Infections and the Diagnosis of Parasitic Diseases. The next five sections discuss respectively the Protozoa, the Nemathelminthes, the Cestoidea, the Trematoda and Arthropoda. The final section is a technical supplement on diagnosis and treatment and is one of the most useful and up-to-date discussions in the book. There is a bibliography and both author and subject indices. The presentation is clear, and the volume is attractively printed and bound. There are a few minor flaws. Some of the maps show greater endemic areas than are commonly accepted, for example the one illustrating the extent of malaria. The fact that specific directions for treatment are in the last section rather than in the discussion of the particular infection where the treatment is indicated may entail additional search for information by the reader. On the whole, this textbook is a valuable addition to the present available literature on parasitic infections and should be widely used by physicians and other workers in this field.

ELIZABETH GAMBRELL.

Dept. of Pathology and Bacteriology
Emory University, Ga.

MANUAL OF CLINICAL CHEMISTRY. By MIRIAM REIMER, M. Sc., Assistant Chemist to The Mount Sinai Hospital, New York. Introduction by HARRY SOBOTKA, Ph. D., Chemist to The Mount Sinai Hospital, New York. 282 pages, with 18 illustrations. New York: Interscience Publishers, Inc., 1941.

In most laboratory manuals, we have always scanned several pages before assimilating a step by step procedure for a chemical analysis. The chief merit of this manual is the brief and concise manner in which the methods are presented. One could tell by a glance at the book that its author is a woman because it is written in the style of a good cook book.

In the preface the author states that this book started with a few directions for internes who were called upon to perform blood sugar, urea, carbon dioxide content as emergency procedures, usually in the middle of the night. The brief manner of presenting the methods was found so convenient and time saving that other determinations carried out in the laboratory were written in the same form.

The introduction explains biochemistry as that which bridges the gap between medicine and chemistry. It goes farther to describe the progress of biochemistry from the time of J. L. W. Thudichum, perhaps the first biochemist to correlate analytic results with clinical manifestations.

The chapter on General Remarks gives detailed explanations on the care of laboratory equipment, such as the cleaning and checking of it. A very comprehensive yet concise table on measures and specifications is given. In addition to that, several pages are devoted to Solutions—kinds, definitions, and directions for preparing.

A most helpful section is that on Synopsis of Blood Constituents. It lists the amount of blood necessary for each particular test, normal range of results and the pathologic conditions correlated with abnormal findings.

Part II of the book is Blood Analysis which includes the Carbohydrates, Nitrogenous Constituents, Inorganic Constituents, Lipids, Pigments, Serum Enzymes, and Blood Gases. For each method given there are directions for all needed reagents, followed by the preferred procedure, standard and calculations.

Chemotherapy forms Part III, including determinations of sulfanilamide in the blood and urine and also a quantitative determination of arsenic.

An extensive section is given on Urine and its Carbohydrates, Nitrogenous Constituents and also several miscellaneous tests. Various tests for complete analysis of cerebrospinal fluid are explained in detail.

The chapter on Feces gives the Guaiac and Benzidine Tests for occult blood. Methods for fat determination, urobilin, and calcium are stated simply, but thoroughly.

Gastric Analysis and its explanations constitute an excellent chapter. The various test meals are given in addition to the routine tests for analysis

One of the most valuable sections is that on Function Tests. Three tests are listed for kidney function and seven tests for liver function. There is also a miscellaneous group covering five tests.

Besides the topics already mentioned the subjects of Sex Hormones, Vitamins and also a miscellaneous section are briefly discussed.

All in all, the reviewer feels that no laboratory should be without this manual.

PLASTIC SURGERY OF THE BREAST AND ABDOMINAL WALL. By MAX THOREK, M. D. 446 pages, with 458 illustrations. Price, $16.50. Springfield, Ill.: Charles C Thomas, 1942.

This is a beautifully gotten up book and it is beautifully illustrated. The male breast comes in for the amount of attention that it deserves. The author stresses that deformity of the breast is a professional handicap to models and dancers, and sometimes a source of great distress to other women. He does not have to work hard to show that gynecomastia is tough for a young man. He believes that all such persons are thoroughly entitled to surgical correction of their deformity. With this in mind he describes a shoal of operations that have been devised for such correction and gives his opinion as to the merits and shortcomings of each.

And yet one wonders why this book was published in 1942.

L. M. B.

ESSENTIALS OF PATHOLOGY. By LAWRENCE W. SMITH, M. D., Professor of Pathology, Temple University School of Medicine; Formerly Assistant Professor of Pathology, Harvard Medical College, and Associate Professor of Pathology, Cornell University Medical School, and EDWIN S. GAULT, M. D., Associate Professor of Pathology, Temple University School of Medicine. With a Foreword by JAMES EWING, M. D., Memorial Hospital, New York City. Second Edition. 904 pages, with 679 illustrations, including 13 color plates. Price, $10. New York: D. Appleton Century Company, 1942.

The second edition of Smith and Gault presents a considerable improvement over the first. Major changes include a more thorough discussion of the fundamental aspects of pathology which was somewhat neglected in the first edition. This change has been made mainly by reduction of the amount of material presented in the form of case histories, and is a decided improvement.

This book differs from the average work in pathology because of its cor relation between pathologic findings and clinical symptoms. Nearly all of the important pathologic states are illustrated by a rather detailed clinical case record. This is a commendable and desirable approach to the subject of pathology since one of its failures in the past has been a lack of correlation between pathologic findings and the clinical phases of disease

The arrangement of the book is also characterized by a distinctive style, in that first there is a general discussion of the pathologic topic, this being followed by an illustrative case, and this in turn followed by a gross and microscopic description of the pathologic process. There is then found a blank page which is inserted for the purpose of taking notes

The illustrations in this book are unusually good, including not only the color plates but the black and white illustrations as well. Furthermore, they have been carefully chosen. On the whole the drawings of blood cells are fairly well done although we may take exception to some of the terminology that is employed. Some of the cells designated as myeloblasts are shown with rather heavy granulation and this reviewer would classify such cells as myelocytes. Furthermore, the cells that are designated as myelocytes could be classified as being more likely juveniles and even band forms. There is an exceptionally good color plate on the arterial diseases.

Altogether this volume appears to be an excellent work in pathology. It is particularly valuable for reference purposes. I do not believe that it fills the need of medical students since it is not sufficiently comprehensive in teaching the fundamentals of pathology. For the more advanced student of pathology it is an excellent work.

After all, pathology is a large subject and it becomes increasingly difficult to present it adequately in a single volume. As a reference work for the general practitioner of medicine I can not think of a better volume available at this time.

ROY R. KRACKE, M. D

WAR MEDICINE: *A Symposium*. Editor, WINFIELD SCOTT PUGH, M. D., Commander (M. C.) U. S. N. Retired, Formerly Surgeon, City Hospital, New York. Associate Editor, EDWARD PODOLSKY, M. D. Technical Editor, DAGOBERT D. RUNES, Ph. D. 565 pages. Illustrated. Price $7.50. New York: Philosophical Library, 1942.

War Medicine is a collection of articles that have appeared in medical journals during the past several years upon the different aspects of injuries which were met with in war surgery, as well as a discussion of aviation medicine. Of necessity there is a great amount of repetition and overlapping. It affords to the surgeon who is interested in war medicine an opportunity to review the literature in one volume. The chief virtue of the book lies in that fact and in its timeliness.

The print is in large type which is easily readable. It is well illustrated.

JOHN W. TURNER, M. D.

THE BIOLOGY OF THE NEGRO. By JULIAN HERMAN LEWIS, Ph. D., M. D., Associate Professor of Pathology, University of Chicago; Member of the Otho Surgical Association, Sprague Memorial Institute for Medical Research; Senior Attending Pathologist, Provident Hospital, Chicago, Illinois. 433 pages. Price, $5. Chicago: The University of Chicago Press, 1942.

One of the Southerner's most deeply rooted beliefs is that he "understands the Negro"—and indeed just naturally knows more about Negroes than anyone could who lives North of the famous line. Of no Southerners is this more true than of the doctors. It may be therefore that this new book from the University of Chicago will receive less attention than it deserves—although much of it is abstracted from Southern sources. Yet it must be insisted that Dr. Lewis has done a scholarly job and he has analyzed a wealth of statistics from all over the world. At the same time he has showed the paucity of reliable statistics as to many aspects of the problem. He corroborates our impression that heart disease, syphilis and tuberculosis are the greatest enemies of the Negroes and notes their assets as fertility, physical stamina, resistance to malaria and certain skin diseases, and ability to undergo surgical procedures. This book should prove a valuable work of reference, particularly to the man who wishes to report statistics on the colored race.

Since this work comes from a University Press however, one is allowed to be hypercritical. One can swallow the split infinitives and an occasional typographical error; it would be easier to accept "data" as a singular noun if it were not also used as a plural; but when Dr. Lewis says (p. 43) "hypothecated"—a word which means "mortgaged"—when he evidently meant "hypothesized" we were all but stalled.

L. M. B.